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CONTENTS.

PAGE

The Etiology of Beri-Beri. Inaugural Address of Session 1901-1902. By PATRICK MANSON, C.M.G., F.R.S., M.D., F.R.C.P., LL.D., *President* 1

Dysentery in Asylums. By F. W. MOTT, M.D., F.R.S. 18

Note by Dr. BAILEY and Dr. BAKER, of Hanwell 45

Discussion on Dr. Mott's paper by Dr. CLAYE SHAW, Dr. PERCY BAILEY, Colonel MACLEOD, Dr. E. W. GOODALL, Dr. OWEN RICHARDS, Dr. WASHBOURN, C.M.G., Dr. ROLLESTON, Dr. T. DUNLOP, Dr. E. J. F. HARDENBERG, Colonel DAVID BRUCE, R.A.M.C., and Dr. PATRICK MANSON 57

The Supposed Infectivity of Desquamation in Scarlet Fever. By C. KILLICK MILLARD, M.D., D.Sc., Medical Officer of Health and Medical Superintendent of the Borough Isolation Hospital, Leicester; late Medical Superintendent, City Fever Hospital, Birmingham 74

Infantile Mortality in the Tropics. By Dr. DANIELS, Medical Superintendent of the London School of Tropical Medicine... ... 90

Small-Pox Hospitals and the Spread of Infection. By JOHN C. THRESH M.D., D.Sc., D.P.H.; Lecturer on Public Health, London Hospital Medical College; Medical Officer of Health, Essex County Council 101

A Doubtful Case of Hæmorrhagic Small-Pox. By Mr. SHIRLEY MURPHY and Dr. KLEIN, F.R.S. 121

An Outbreak of Syphilis in an Indigenous Tribe in India. By LEONARD ROGERS, M.D., M.R.C.P., I.M.S., Officiating Professor of Pathology, Medical College of Calcutta 129

Filariasis in the West Indies and its Prevention. By GEORGE C. LOW, M.A., M.B., C.M. (Edin.); Craggs Scholar; London School of Tropical Medicine 134

Obituary Notice :-

John Wichenford Washbourn, C.M.G. 151

135729

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1868. Guipon, D. J., M.D., Laon, Aisne, France.
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1888. Koch, R., M.D., 36, Klosterstrasse, Berlin.
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TRANSACTIONS

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The Etiology of Beri-Beri.

By PATRICK MANSON, C.M.G., F.R.S., M.D., F.R.C.P., LL.D.,
President.

(*Read : November 15th*, 1901.)

Gentlemen,—The last two or three decades have been signalised by many important discoveries in the etiology of disease: of tropical diseases no less than of the more familiar diseases of temperate climates. In the group of tropical diseases we may confidently tick off as having had their exact causes finally determined malaria, Mediterranean fever, plague, leprosy, filariasis, ankylostomiasis, endemic haematuria, endemic hemoptysis, mycetoma, and some minor complaints. Quite recently, thanks to the insight of some of our American confrères, we are almost justified in adding yellow fever to the list, although it is true the actual germ of this disease has not been definitely isolated. The immense practical gains which have already sprung, or which will ultimately spring, from these discoveries are patent to everyone. Their scientific importance is in many instances of the first magnitude. These considerations, utilitarian and scientific, should stimulate to further effort towards attempting the solution of the many etiological puzzles which, more especially in tropical diseases, still remain unsolved.

Among other tropical diseases, the various affections whose leading symptom is intestinal flux, and which are included under the somewhat elastic names of dysentery, sprue, chronic diarrhœa, colitis, entero-colitis, etc., are, perhaps, the more important of the unsolved etiological problems. Although intestinal flux is one of the commonest symptoms in disease, especially in tropical disease, strange to say the cause of the pathological processes which lead to the symptoms are, if we except cholera, absolutely unknown; or, at all events, undetermined.

Perhaps only second in importance and in mystery to the intestinal fluxes, are those tropical affections which have in common as their leading pathological feature multiple peripheral neuritis. It is about the etiology of one of these, namely, beri-beri, that I propose to offer a few observations this evening.

Of the importance of beri-beri it is hardly necessary to speak. Everyone familiar with the literature dealing with the diseases of the East is aware of the almost leading place which, in certain localities and seasons, beri-beri assumes in their pathology. Instances in illustration of this can readily be adduced from Japan, the Philippines, China, Tonkin, the Netherlands Indies, Burma, the Coast of India, tropical Africa, the Brazils, West Indies, Sandwich Islands, and even Australia. Among British possessions nowhere is the disease more common than in the flourishing Straits Settlements, and in the neighbouring and intimately associated Federated Malay States. Here it is always in evidence. A large proportion of the beds in the State and other hospitals are occupied by the victims of beri-beri. Thus, in the State of Negri Sembilan, Dr. Braddon reports that in the year 1899, out of every 1,000 deaths of Chinese immigrants, 62 were attributed to beri-beri. Of every 1,000 deaths of the native Malays 140, and every 1,000 deaths among Tamils 150, were caused by this disease. So that about one-tenth part of the entire mortality of that State was attributable to beri-beri. During the nine years 1890-1899, in a mean population of 80,228, 6,001 cases of beri-beri were treated in the hospitals; of these 535, or 9.2 per cent., died.

In the year to which I refer, 1899, according to Dr. McClosky, in Pahang, in a population of 73,000 there were 146 deaths from beri-beri. On the assumption that the case-mortality in Pahang was similar to that of Negri Sembilan, this would represent a total of 1460 cases, or about 2 per cent. of the entire population. In Berak, also in 1899, according to Dr. Wright, in an estimated population of 294,297, there were treated in the hospitals 3,113 cases of beri-beri, with a mortality of 333. In the same year, 1,793 cases were treated in the hospitals of the Straits Settlements, with a death-rate a little over 32 per cent.; a death-rate, be it remarked, much greater than that occurring in the Malay States: a circumstance attributable in some measure probably to the fact that only graver cases were admitted.

It would be easy to bring together similar statistics

from other countries. The figures I give have been selected because they are recent, apply to British territories, and have been collected by responsible medical officers. They show quite conclusively that, in some parts at least of our Empire, beri-beri is a very important element in the State problems which the Government has to tackle. Directly or indirectly, it is a serious tax on these communities, and a distinct drag on their progress. Not only does it lead to great loss of life, but it is an enormous drain on the labour market and on the industrial resources; for beri-beri is a disease which, when not quickly fatal, usually runs a long course—probably of several months—during which the patient is, as a rule incapable of earning his living; and, in many instances, of even cooking his food, or in other ways attending to his personal requirements. It hampers every industry. It breaks out constantly on plantations, in mines, schools, gaols, and hospitals, and is a source of continual anxiety to the capitalist, the employer of labour, the Government official, and the medical men. To be able to place one's finger with precision on the cause, or on the medium or way in which it is conveyed, would probably be to enable us to stop this disease, and so confer an incalculable boon on the countries I have enumerated.

The etiology of beri-beri, therefore, is well worth the serious consideration of the sanitarian as well as of the epidemiologist. As the subject is to occupy the attention of the Commission which, as suggested in my Address last year, should be sent out to study the diseases of the South Pacific (a Commission which, I am pleased to be able to inform you, has already set out), I have thought that the present would be an appropriate opportunity to pass in review some of the facts which have been accumulated and theories which have been formulated in connection with the subject.

An additional consideration which has influenced me in selecting the etiology of beri-beri as the subject for my Address, and one which should urge us to fresh effort at solving the mystery of this disease, lies in the circumstance that there is a considerable body of evidence tending to show that beri-beri has spread into regions hitherto believed to be immune, and even into Europe and other temperate climes. As a matter of fact, beri-beri is constantly to be found in the shipping in the London Docks; and, doubtless, if carefully sought for, it could be found in all the large shipping ports, not only

in Britain, but on the Continent and North America. Moreover, beri-beri, or a disease closely resembling it, if not actually identical, has shown itself in more than one institution in this country—in the Richmond Asylum, for example. A similar affection has shown itself elsewhere in Europe—in France and Italy, for example—and also in the United States of America. It is quite possible, therefore, that in time an epidemic disease resembling beri-beri, if it be not true beri-beri, may attain a permanent lodgment in temperate climates, and thus add another burden to countries already sufficiently afflicted with their own indigenous diseases. In these days of rapid and increased communication, all kinds of diseases tend to become diffused.

The study of tropical disease, as compared to the study of the diseases of Europe, labours under this disadvantage: that whereas in the latter there is in most instances complete agreement amongst pathologists as to the pathological conditions indicated by certain names, in the case of tropical diseases, owing to the physical difficulties imposed by distance, and consequent rarity of opportunity for the comparison and identification of the diseases of places it may be thousands of miles apart, much confusion has crept into the nomenclature. In some places, a disease receives a name which in other places is applied to a totally different condition. Thus, as regards the disease I am discussing, the term "beri-beri" was applied in Assam and Ceylon to what turns out to be Anchylostomiasis, a condition widely different from the peripheral neuritis which the term "beri-beri" indicates in Brazil, Japan, Netherland Indies, and elsewhere. Furthermore, it is just possible that the term "beri-beri" includes more than one species of peripheral neuritis. It has even been suggested that what is known as "beri-beri" is, in many instances, if not in all, a metallic neuritis, produced by arsenic or other mineral.

In the tropics, as in temperate climates, there are undoubtedly several distinct kinds of the pathological condition passing under the name of multiple peripheral neuritis. For example, I have seen cases of this condition from the West Coast of Africa, which, although bearing a close resemblance to beri-beri, were probably of an entirely different nature. At all events, the cases I refer to did not exhibit the cardiac and dropsical symptoms usually present in true tropical beri-beri. I might remark in passing that there is a large and almost unworked field waiting for the neurologist in the Tropics; for example, the type of neuritis

I refer to, as well as that singular condition so common in Europeans in West Africa, and known as "West Coast Memory."

One well-marked type of peripheral neuritis has been very carefully described by Dr. Strachan. In many respects it, too, resembles beri-beri. Dr. Strachan describes a typical case thus: "A patient presents himself, complaining of numbness and cramp in his hands and feet, dimness of sight, and a tightness round the waist." This is always the remark first made by the applicant for treatment. If the case be somewhat more advanced, he may add to this statement that he fears he is "getting hard of hearing;" and he goes on to say that he suffers from severe burning in the palms of the hands and soles of the feet, and that very often this is worse at night than in the day, and that the pains and the burning heat prevent his resting. It is rarely that he gives more information than this. On examination, it will be seen that there is slight excoriation with fine branny desquamation of the edges of the eyelids, margins of the lips, and around the margins of the nostrils. The palpebral conjunctiva may be hyperæmic as well as the lips. The heat in the hands complained of by the patient will be found to be not merely subjective, but appreciable to the touch, and due to a hyperæmic condition of the palms. The acuteness of vision for form will be found to be more or less impaired, according to the stage to which the malady has progressed. Examination of the main nerves to the extremities will show that they are very tender on pressure, especially the ulnar nerve; and along the distribution of their terminal filaments they may be tracked by fine herpetic vesicles. On admitting such a case to hospital, and watching its further progress, it will be noted that at night the patient will be awake for hours, rubbing his feet and legs most probably, and moaning with pain. The loss of vision will proceed until he can with difficulty distinguish a large object immediately in front of him, and cannot recognise individuals. The muscles of his limbs will waste until the claw hand and foot are marked features; and this wasting of muscles and disappearance of fat will produce an emaciation which is very noteworthy in advanced cases. There will be found to be no alteration of the pupil to light and accommodation, no falling when the eyes are closed, and the sphincters will not be affected. Should the disease make further headway, the patient may become a mere helpless skeleton, unable even to feed himself, his breathing laboured from implication of trunk

muscles in the general muscular atrophy. Almost blind, and with perhaps an ulcer on the cornea, quite deaf, and with possibly small bullœ on the extremities. There may also occur, during the course of the malady, monoplegiæ, as facial palsy, or some of the external muscles of the eyeball, and, but rarely, of some group of muscles in an extremity. The temperature chart will show a sub-normal condition in the mornings, with an evening rise of from 1 to 2 degrees. A fatal termination is fortunately rare: when it occurs it is due to the dyspnœa and the riotous action of the heart, resulting from vitally important nerves, becoming involved in the now almost universal nerve changes. As a rule, however, under appropriate treatment, recovery gradually—with perhaps from time to time slight recurrences of the nerve inflammation—takes place. The patient becomes stronger, can help himself a little, assimilates food well, and puts on fat again. Then he is able to walk a little, first with help, and afterwards alone; his grasp, measured with the dynamometer, shows daily increase of muscular power, his sight clears up, and his deafness gradually passes away. Though, if this has been extreme, it is usually one of the last symptoms to disappear." This disease attacks hundreds of negroes in Jamaica, at all ages and of both sexes. It prevails most on the sea-coast and in low-lying regions. Dr. Strachan is inclined to attribute the affection to malaria; but he does not pledge himself to this opinion, and I think wisely: for, were this a malarial neuritis, a similar disease ought to be found in other malarial countries. In many respects Strachan's disease resembles arsenical neuritis, and is well worth further investigation in the light of the recent experiences in Birmingham and the North of England.

Another curious neurosis, also probably a peripheral neuritis, was noticed by Malcolmson and other writers on East Indian disease in the earlier part of the last century; I refer to what is known as "Burning Feet." This name adequately describes the leading symptom of the disease, for such undoubtedly it is. It was, and probably still is, very common in parts of India. It occurs in China to my knowledge.

It is evident, therefore, in investigating and discussing the etiology of beri-beri, if confusion is to be avoided, that a clear conception of what is meant by this term should be arrived at. What I conceive to be understood by the word "beri-beri" is a form of multiple peripheral neuritis which occurs endemically and epidemically,

and is specially characterised as compared to other forms of peripheral neuritis by proneness to œdema and to implication of the neuro-muscular system of the central organ of circulation; by complete absence of trophic skin lesions, of paresis of the muscles of the head and neck, of marked implication of the organs of sight, hearing, taste and smell, and of the mental faculties. In common with alcoholic and arsenical neuritis, there are troubles of locomotion, paresthesiæ of various descriptions, especially in the lower extremities, marked hyperesthesia of the muscles involved with, subsequent atrophy; and generally, after the initial stages, complete absence of patellar tendon reflex, the superficial reflexes and the action of the sphincters being in the vast majority preserved. The patient complains principally of weariness, palpitations, and breathlessness, weakness and numbness of the extremities, swelling of the legs. Although, at the commencement of the disease, and at the outset of such exacerbations as may occur during its progress, there may be slight elevations of temperature and gastro-intestinal disturbances, fever and diarrhœa are far from being prominent features in the progress of the complaint. The intensity and duration of the disease varies within wide limits. It may be of the most trifling description and only of a week or two's duration; or it may prove rapidly fatal; or it may persist in varying degrees of intensity for months. The leading feature may be paresis and muscular atrophy, or it may be extreme anasarca, or it may be serous effusion into pericardium or pleura, or it may be œdema of the lungs, or it may be rapidly fatal paralysis of the right heart or of the diaphragm and muscles of respiration. It occurs generally in limited epidemics in particular houses, institutions, plantations, mines, etc.; or it may be spread over a large area, but only attacking limited foci therein. It occurs at sea on board ship, where it is prone to be extremely fatal. The post-mortem lesions are those ordinarily found in multiple peripheral neuritis, inflammation, and degeneration of peripheral nerves, and, as Dr. Wright has recently shown, an ascending degeneration of the neuron, ultimately involving the corresponding intercranial cells. It may prove fatal very rapidly in a day or two from the declaration of the symptoms, or at any time during its progress. One attack confers no protection against a second. The case-mortality ranges from 5 to 50 per cent. In some epidemics only a small proportion of the affected community is attacked; in other epidemics almost every

one may be victimised. In some plantations over 75 per cent. of the coolies have been killed off by beri-beri in a single year.

What is the cause of this disease? Innumerable speculations have been advanced. So far, none of these has been proved to be correct. I shall pass over the theories of the earlier writers on the subject, and confine myself to what may be regarded as the not-disproven theories of more modern workers. These may be grouped under two headings: (1) the dietetic theory; and (2) the microbic theory.

The dietetic theory has been advocated by many writers, especially by Miura, who attributed beri-beri to the use of fish. A more plausible hypothesis has been advanced by Vinson, Le Roy de Mericourt, Rochard, Overback de Meyer, Mesé, and especially by Takaki, all of whom attribute the disease to a prolonged and uniform rice diet. Takaki, in particular, believing that the physiological deficiency of nitrogen in the almost exclusively rice diet, which at one time prevailed in the beri-beri-stricken Japanese Navy, was the cause of the disease, urged on the Japanese authorities the adoption of a more nitrogenous diet for their sailors; with the result that, whereas before the adoption of the more liberal dietary 32.45 per cent. of the force were annually attacked with beri-beri, after the adoption of the new dietary in 1884 the incidence of the disease rapidly fell to zero. In 1898 it stood in a force of 18,426 men at 0.08 per cent. only. So remarkable a result as this could not fail to call for the application of the principle involved to the army, to prisoners, and to similar public bodies; and, as stated in the *Sei-i-Kwai Medical Journal*, by Baron Saneyoshi, F.R.C.S. Eng., with equally happy results. Moreover, it has been observed that wherever throughout the Japanese Empire, from one circumstance or another, the diet of the people has been exclusively or mainly a rice one, beri-beri has appeared, to disappear again when this assumed dietetic error had been corrected.

This remarkable experience certainly tends to support Takaki's views; but if we consider the circumstances of certain epidemics elsewhere it becomes manifest that there is something more than—or rather, other than—nitrogen starvation at the root of beri-beri; that very probably nitrogen starvation has nothing to do with it whatever. Takaki's theory may have led to a successful practice; but success in practice does not necessarily prove the correct-

ness of the theory on which the practice is founded. If the nitrogen starvation theory be correct, how comes it that beri-beri not infrequently attacks the well-to-do? That it appears in hospitals and gaols, where the food is of good quality, physiological in the proportion of its elements, and sufficient in amount? For example, in the Tan tok seng Hospital, Singapore, the diet is a liberal one; nevertheless patients admitted for some trifling disease, such as an ulcer of the leg, after a time may contract and die from beri-beri. How comes it that the disease appears in gaols, schools, plantations, and mines, the dietaries of which have been specially devised with a view to avoiding deficiency in the nitrogenous elements? Many examples of this could be brought forward. In 1894, a number of Japanese were imported into Fiji to work on sugar estates. They were picked coolies, and in good health when they left Japan and during the journey. On their arrival to Fiji they were divided into two bands; one, consisting of 50 coolies, was sent to work on a certain estate. The dietary was liberal, including meat or fresh fish to the amount of half a pound *per diem*, besides two pounds of rice, and a variety of condiments. Nevertheless within two months of the arrival of the coolies in the island beri-beri broke out, and in all 42 cases occurred. Eight of these died; 34 were returned to Japan suffering from the disease, the remaining eight healthy coolies accompanying them. The other batch of immigrants, 205 in number, were sent to a sugar estate in another part of the island. They had a similar dietary. Within a month beri-beri broke out among them. The cases steadily increased in number, and by the end of six months 226 out of the 250 were affected. Sixty-nine died. The survivors, 181 in number, many still affected with beri-beri, were sent back to Japan. In Singapore Gaol, in 1898 and 1899, there were two kinds of diet in use: ordinary and penal. The ordinary diet was a sufficiently liberal one, including fresh meat and wheaten flour. The penal diet contained no meat. Nevertheless the greater number, absolute and proportionate, of cases of beri-beri occurred among these on ordinary diet—that is on the diet richest in nitrogen.

Macleod* relates an interesting epidemic on board ship, in which beri-beri attacked the captain and officers, and not the crew—an unusual occurrence. The dietary of these officers included such luxuries as haddies, clams,

* *Brit. Med. Jour.*, August 14th, 1897.

oysters, lobster and salmon. Here there could have been no nitrogen starvation. The disease elected to attack the better-fed members of the ship's company. There was no nitrogen starvation among the inmates of the Richmond Asylum before or during the epidemics of beri-beri there, so fully and carefully described by Dr. Conolly Norman. There was no nitrogen starvation in the Blind Asylum at Rio de Janiero, where not only the inmates, but the director and his family were attacked.

There are many additional instances on record, and which might be quoted, in which Europeans enjoying a liberal diet have fallen victims to beri-beri; and although it must be admitted that remarkable improvement has sometimes supervened on the introduction of a more liberal dietary in beri-beri smitten jails, and similar institutions, yet even in those same places, and while the inmates were still enjoying the improved dietary, which at one time seemed to be beneficial, the disease, after a year or more, reappeared. This has been the experience in the Singapore Gaol, where the disease reappeared in 1898, after having been absent for 14 years.

If the cause of beri-beri be deficient nitrogen in food, we ought always to find this condition preceding or accompanying its epidemic development, and where we get nitrogen starvation we ought always to get beri-beri. How far the first is from being the case, the instances I have quoted show; how far the second is from being the case, hundreds of unintentional experiments daily prove: for, surely, nitrogen starvation is common enough the world over; beri-beri has no such universal distribution.

F. C. Eijkman, of Batavia, has recently brought out a curious fact in relation to rice diet in beri-beri. The prisoners in Java are fed, amongst other things, on three qualities of rice—one, decorticated rice, in which the pericarp has been entirely removed, or at least in 75 per cent. and upwards of the grains, by milling. A second kind, in which the pericarp is retained entirely, or at least in 75 per cent. of the grains; and a third kind, consisting of a mixture of the first and second. Statistics applying to 280,000 prisoners show that beri-beri attacks 2.84 per cent., or 1 in 39 of the prisoners who are supplied with the first, or decorticated rice, whilst it attacks only 0.01 per cent., or 1 in 10,000 of the prisoners who consume non-decorticated rice; those fed on the mixture of these two qualities suffer in an intermediate degree, namely, 0.24 per cent., or 1 in 416. He further states that fowls fed exclu-

sively on decorticated rice are affected with a peculiar paretic disease resembling beri-beri; whereas fowls fed on non-decorticated rice are not so affected. The source and species of rice, Eijkman says, makes no difference in the result. Other observers in the Malay Islands have made somewhat similar observations on the influence of the quality of the rice. The Japanese substitute barley or wheat for rice as a preventive of beri-beri, and believe in their efficacy. A grave outbreak of beri-beri in the Pearling fleet, off the north coast of Australia, was arrested, it is said, by the timely substitution of wheaten flour for the damaged rice on which the crews were feeding. These are facts to be noted, but not necessarily interpreted as proofs that beri-beri is a rice-caused disease, much less a nitrogen starvation disease.

The Germ Theory of Beri-beri.—Another theory, and one much more plausible than the foregoing, is to the effect that beri-beri is a germ disease. Such a theory is quite compatible with the remarkable Japanese experiences to which I have already alluded, as well as with all the known facts. But as to whether the germ produces its morbid effects while proliferating in the human body, or as to whether it acts indirectly by producing outside the body a toxin which, on being ingested, inspired or otherwise absorbed, acts on the nerves, it is impossible as yet to say. There are arguments in favour of both hypotheses.

The fundamental nerve lesions in beri-beri resemble those produced by several well-known organic poisons, and are probably the results of the direct action of some such poison. If we adopt the first hypothesis, that is the infection hypothesis, we have the argument supplied by analogy for supposing that the lesions are the effect of a toxin elaborated by the germ while it is proliferating in the blood or tissues, that is to say, an intoxication produced by an infection. If, on the other hand, we adopt the second hypothesis, we must conclude that the specific germ resides in some external medium where it elaborates its toxin which is subsequently absorbed as such, the germ not entering the body, or, at all events, not necessarily so.

(*a*) The infection theory has had many advocates, including Scheube, Lacerda, Ogata, Masanori, Wallace Taylor, Cornelissen, Suguenoya, Pekelharing, and Winkler, Van Eecke, Rebourgeon, Musso, and Morelli, Glogner, Braddon, Hunter, Rost, and I do not know how many more. Many of these claim to have discovered a beri-beri bacterium.

Unfortunately, in some instances the observations are crude and open to objection on the ground of technique; more unfortunate than this, there is a suggestive want of uniformity as to the morphological and cultural characters of the various organisms separated, some describing a bacillus, others a micrococcus, one at least an endocorpuscular protozoon like that of malaria. There is also a suspicious unanimity as to the pathological effects produced by those very different organisms when injected into the lower animals. Some recent work by Hunter, in Glasgow, carried on under very favourable circumstances, led him to the conclusion that beri-beri is caused by a micrococcus which is visible in the blood, and which can be cultivated in various media, and on subsequent injection into rabbits give rise to something like a peripheral neuritis. His observations, like those of Van Eecke and Braddon, seem to confirm those of Pekelharing and Winkler. On the other hand, equally careful observations, made in equally favourable circumstances by more than one bacteriologist, working with material supplied by the Richmond Asylum epidemic, gave absolutely negative results; the same may be said of similar observations elsewhere. If beri-beri be an infection, I hold with Scheube—than whom there is no higher authority on this disease—that so far the germ has not been discovered, or, at all events, that no germ has been demonstrated to be the specific cause of the disease.

To my mind, the theory which conforms best to all the known facts in respect to the etiology and pathology of beri-beri is to the effect that this disease is purely an intoxication produced by a toxine elaborated by a germ whose nidus is located outside the human body; that in this respect beri-beri is on all-fours with alcoholism, the germ of which is the yeast plant, the nidus solutions of sugar, the toxine-alcohol, and, to complete the parallel, the pathological effects—a peripheral neuritis.

There is no evidence to show conclusively that beri-beri can pass directly from men to men like the ordinary infectious diseases; yet that it is produced by a living germ is certain. This is proved by the facts (*a*) that the cause can be transported from place to place, and therefore cannot be of a climatic or meteorological nature; and (*b*) that when so transported it can multiply and spread, and therefore cannot be of an inorganic nature. As already mentioned, it has recently been suggested that beri-beri is caused by arsenic. Now the clinical symptoms of beri-beri and arsenical poisoning are very different. I have

never seen skin lesion, other than accidental, in beri-beri. This in itself is almost conclusive. The diseases are not the same clinically. Professor Dixon Mann has recently shown that in arsenical neuritis arsenic can by chemical tests be detected in the hair. Some time ago he had the kindness to examine for me hair from two recent cases of beri-beri. In neither did he find a trace of the mineral. There is no evidence worth considering that tends to show that any other mineral is concerned in the production of the disease. Tin has been suggested; but there is no tin in Japan, or in the Kurile Islands, where beri-beri has occurred. A mineral cause would not be regulated in its incidence or effects by meteorological conditions, as beri-beri certainly is.

That the cause of beri-beri can be transported has been proved over and over again, as, for example, by the epidemic among Japanese in Fiji already alluded to; but it is more difficult to find facts to establish completely that when so transported it can multiply. If, however, we find an instance of some island hitherto free from the disease, among the natives of which beri-beri, on being introduced, has spread, we may regard it as presumptive proof that the cause of beri-beri is endowed with that special property of living things, the power to multiply. There is recorded at least one apparent example of such an occurrence. A number of Tonkinese and Annamese were brought to New Caledonia in March, 1891. Beri-beri broke out among them and caused many deaths. After a time the disease spread and in some instances proved fatal to certain natives, Kanakas, who had become associated with the Asiatics. These two things, portability and the power of multiplication, if established, certainly show that the fundamental cause of beri-beri is a living thing, a germ. The evidence that the multiplication of this hypothetical germ occurs outside the body, though not conclusive, is certainly stronger than any evidence hitherto advanced in favour of its multiplication inside the human body. Did the germ multiply inside the human body we might expect that the disease it produces would run a more or less definite course, as in other infections. But this is not so: for, if we remove the subject of beri-beri from the infected area or the conditions in which he sickened, after an interval of from a week to a fortnight the progress of the disease, if it had not proved fatal in the interval, will be arrested and slow convalescence supervene. Now, if beri-beri be caused by a germ living in the human tissues, it is not likely

that this germ would be killed off thus early; on the contrary, analogy would lead us to believe that the germ would continue to multiply till immunity had been produced; and this we know to be a long process in beri-beri, for those patients whose lives are spared and who continue to live in the endemic area generally exhibit active symptoms for months after the commencement of the disease.

In our experience of the disease at the Seamen's Hospital, Albert Docks, we have very strong evidence of this evanescent nature of the primary effects of the toxine of beri-beri. From October, 1890, to November, 1901, 135 cases of beri-beri have been admitted to this hospital. They all came direct from the ships in which they acquired the disease. 19 died; 4 on the first, 2 on the third, 4 on the fifth, 2 on the sixth, 1 on the eighth, and 3 on the fourteenth day after admission. This accounts for 17 of the 19 deaths. Of the two other fatal cases, one died from phthisis on the 104th day after admission, and the other, apparently from dilated heart, on the thirty-first day. With the exception of these two, neither of whom apparently succumbed to the immediate effects of disease, a fatal result never occurred later than a fortnight after the patient had been removed from the conditions in which he had contracted the disease. At the Seamen's Hospital we therefore have come to regard cases of beri-beri as safe when they have passed the fourteenth day. Would this be justifiable were the disease the result of an infection, immunity from which is only slowly acquired. Beri-berics when kept under beri-beri influences do not recover for months, during which the disease is liable to exacerbations in any of which death may occur. Beri-beri is so like alcoholism in these respects—keep the drunkard from liquor, in a few days he begins to recover; give him liquor, and he will not recover; take the beri-berie away from the circumstances under which he is being poisoned, in a few days he begins to mend; keep him under these circumstances, and he will continue sick and very likely die.

Pekelharing and Winkler, who advocate the personal infection theory, try to get over the generally-recognised beneficial influence of removal from the endemic area by assuming that in this area the disease is kept up by continual or repeated infections; and in their experiments they attempted to imitate what they assumed occurs in nature by introducing syringefuls of their cultures at short intervals into the experimental rabbits. Their followers have done the same. Small wonder they had fatal results,

and indications of peripheral neuritis; it is a wonder, rather, that any of their animals survived such treatment. Hirota publishes an interesting observation which lends strong support to the toxin theory. In some respects it resembles our experience at the Albert Dock Hospital. Infants suckled by beri-beri get the disease. He refers to fifty-two such cases. When the infants were weaned and fed on condensed or cows' milk improvement set in at once; five cases which were not so treated died. A child who acquires a germ disease from its mother's milk will not recover thus rapidly; but a child who has only become intoxicated from drinking an intoxicated mother's milk will.

But although we may conclude with a fair show of reason that beri-beri is a toxine-produced disease, we cannot say what the toxine is, what the germ producing it is, or where this germ resides. The whole epidemiology of the disease shows that the germ clings to people—witness its transportation to Fiji and New Caledonia by Asiatics, and to places, witness the persistency of the disease in certain buildings and ships. Instances of its tendency to cling to buildings are numerous, and there are some well-authenticated examples of a similar persistency in ships. A very telling example of the latter is recorded by Mr. Montgomery Smith, in the *British Medical Journal* of November 5th, 1898. In 1896 a ship, the *Lodestar* of London, arrived in Falmouth with a cargo of rice from Rangoon, after a long voyage, during which the entire crew was attacked with beri-beri, and three had died from the disease. The ship was sold to German owners, and re-named *Steinbek*. At Amsterdam she received a new crew, and took in fresh provisions. She then sailed for Java, where she loaded with sugar. She left Java on Jan. 27th, 1898, all being well on board. On April 29th, when off St. Helena, and after being three months at sea, beri-beri broke out, and when the ship arrived at Bermuda the entire crew, with the exception of one man and a boy, were down with the disease. One man died in Bermuda, the others, apparently, recovered. Two epidemics of beri-beri in the same ship, but in different crews and on different provisions, occurring at an interval of about two years, is surely no mere coincidence, but distinctly points to a persistent infection in the ship.

A Chinese revenue cruiser, well found in every respect, visited Corea. Many of the crew subsequently developed beri-beri. For several years thereafter, during the south-

west monsoon, cases of the disease kept cropping up in the crew. This example of ship infection came under my own cognisance, and I was familiar with the facts.

At the Albert Dock Hospital, as recorded by Dr. Rees (*British Medical Journal*, Sept., 1898), we came at one time to regard certain steamers trading to the port of London as infected ships. We called them beri-beri ships, as they almost invariably brought us cases of the disease on their return from their periodical voyages to the East.

Many similar facts could be adduced tending to show, although not absolutely to prove, that a ship can become infected with beri-beri. If a ship can become infected, why not the houses, and even the localities in which this disease, as is its habit, establishes itself? Granted that a place or ship can become infected, we have still to find the particular medium in which the germ operates, and the particular portal through which its toxine enters the human body. There is a marked tendency among the students of this subject to regard, not without reason, rice as the nidus of the germ; but, if rice be the medium, how is it that the disease sometimes occurs in individuals who have not consumed this cereal? The answer may be that other farinaceous substances are equally efficacious as culture media, if I may so speak. But if we concede this, how are we to explain the latency of the infection in buildings and ships which have had their stores frequently renewed in the intervals between the epidemics? The answer to this may be, that the germ clings to the walls, the wood, the utensils of the place; and that when cereals are brought in this germ drops into them and infects them. Thus infected, when consumed, whether on the premises or exported elsewhere, they may cause the disease. On the other hand, it may be that the germ distils its toxine from some other medium; it may be clothes, the soil, wood, or what not, and so poison through the atmosphere. This is speculation, but I think we have distinct evidence to show that beri-beri is, in a sense, a place disease.

In the foregoing reasonings—or, if you like, speculations—on the nature or cause of beri-beri, I have been powerfully influenced by the analogy of alcoholism. Analogy, I know, is not argument, but if it helps us to understand, or if it helps to suggest, it is not quite useless. Before, however, anything like assurance on the etiology of beri-beri can be attained, we want more facts; above all, carefully devised and executed experiments. It is to be hoped that the expedition to which I have alluded will, in the specially

favourable circumstances in which it will be placed, carry out a series of such experiments with the view to ascertain: first, the medium or media in or through which the disease is conveyed; and, secondly, having found this, by a process of exclusion to isolate the organism which undoubtedly is at the root of the disease. Should they succeed in this, it is more than probable that we will be in possession of that knowledge which alone can give precision and accuracy to efforts at cure and prevention.

There are several points that investigators in the etiology of beri-beri must be careful to attend to.

1. The diagnosis. They must avoid mistaking other forms of peripheral neuritis for that of beri-beri.

2. They must bear in mind the possibility that the disease may not have been contracted at the place in which it is declared.

3. That the toxine which produces an outbreak of beri-beri may have been imported as such, and not manufactured, so to speak, locally.

4. They must carefully differentiate between predisposing or favouring conditions, such as overcrowding, heat, and moisture, bad food, etc., and the actual direct cause.

Finally, they must recognise that the actual cause must correspond in its geographical distribution with the geographical distribution of the disease.

DYSENTERY IN ASYLUMS.

By F. W. MOTT, M.D., F.R.S.

(*Read: December 13th*, 1901.)

Mr. President,—Sir, some time ago you did me the honour of asking me to read a Paper on "Dysentery in Asylums," in consequence of a Report issued by Dr. Durham and myself on that subject, which, on account of its length and voluminous details, required summarising.

When I was appointed Pathologist to the London County Asylums I soon recognised that dysentery, or, as it was called, "Colitis," was very prevalent at Claybury, and that numbers of people were dying of a preventable disease, which had assumed an epidemic form. So impressed was I with this, that I asked the late Prof. Kanthack to assist me in its investigation. Unfortunately, he left for Cambridge, and shortly after I became the subject of typhoid fever. In the summer of 1898 I met Dr. Durham, who told me that he had been investigating an outbreak of severe bowel complaint which had occurred in an asylum, the authorities of which did not wish publicity. I told him this complaint was very prevalent at Claybury, and probably at many other asylums, if it were known; he therefore came to my laboratory to work at the subject, and the result was a Paper in the Archives of Neurology, 1899, on the "Etiology of Asylum Dysentery," mainly dealing with the question of a specific micro-organism.

In September, 1898, after some opposition, it was agreed that I, the Pathologist, should make the post-mortem examinations at Claybury, and elsewhere if necessary; and since then the bowels have been systematically examined in every case that has died; and in my report, presented May 16th, 1899, I pointed out the large percentage of people that had died with dysenteric lesions at Claybury. The Asylums Committee was thus led to institute an investigation of its cause and remedy, which was subsequently extended to all the London County Asylums; at my request a bacteriologist was appointed, and I have much pleasure in testifying not only to the scientific ability with

which Dr. Durham undertook this task, but also to those other attributes, equally important in such an investigation —courage, honesty, and common sense, all of which were equally shown by the Chairman of the special Sub-Committee appointed to consider the question. We instituted a printed form of notification of all forms of diarrhœa and dysentery, and issued it to all the London County Asylums; the investigation was continued for six months; a report was prepared, submitted to the special Sub-Committee of the Asylums, which was circulated to all the members of the committee and to the superintendents, who were invited to make comments thereon. The Sub-Committee, which included three medical men, decided to recommend the Asylums Committee to print the report in full, and circulate it to every asylum in the country.

Definition of the Disease.—A disease characterised by febrile onset and by inflammatory lesions of the intestine, affecting persons of different ages and bodily health, and tending to occur in outbreaks. As a rule, it is more liable to affect the old, the infirm, and the bedridden; this, however, was not the case at Claybury; but no one particular form of insanity is more liable than another. Occasionally, attendants, nurses, and even the higher officials of the staff, suffer. Besides the great prevalence in the form of outbreaks, the statistics of Claybury Asylum show that the disease is endemic throughout the year.

Nomenclature.—The disease which occurs in lunatic asylums of this country, and which is characterised by certain clinical symptoms and inflammatory anatomical lesions of the large intestine, is designated "Colitis," "Ulcerative Colitis," "Blood and Mucous Diarrhœa," Zymotic Diarrhœa," and "Asylum Colitis or Dysentery." The term "Colitis" is rather a word for use in the post-mortem room, as signifying an anatomical alteration of the colon; and this is not exact, for other parts of the intestine may be affected; we do not use the term "enteritis," or "ulcerative enteritis," but enteric or typhoid. Moreover, it has another objection: it carries no meaning to the lay mind; and thus the authorities who control the administration of asylums are not so readily brought to the sense of the gravity of an epidemic as when the term Dysentery is used. Again, it is not right to use the term "Ulcerative Colitis," for the most severe and acute cases may terminate fatally before there is sufficient time for ulceration to have taken place; moreover, it is highly probable that some

cases that run a mild course may not be associated with ulceration of the large intestine at all. Blood and mucous diarrhœa does not include all cases of the disease. Zymotic diarrhœa indicates its infective nature, but not its leading clinical symptoms. Lastly, the question arises whether the simple term "dysentery" should be used, or whether it should be qualified as Asylum Dysentery or English Dysentery.

The disease which exists in asylums is, to my mind, the same as was described by Sydenham, Willis, Morton, and again by Latham in the Millbank Prison epidemic. It is customary to look upon dysentery now as a tropical disease, yet it is well known that a disease which is practically identical in its clinical and pathological characters occurs more or less throughout Europe, and even in the coldest climates where sanitary conditions are defective.

There may be, as your President, Dr. Manson, suggests, several forms of dysentery. I find that most authorities who have studied the disease in the Tropics consider the *Amoeba Coli* as the essential causative agent; but Flexner and Barker, in the Philippines, did not find the *Amoeba* in the acute cases, only in the chronic. Councilman and Lafleur, in dealing with amoebic dysentery, admit that the disease is an acute inflammation affecting primarily the submucosa, with following necrosis of the mucous layer. That is exactly what Dr. Durham and I have found on microscopical examination of the rapidly fatal acute asylum cases, dying in a few days, or within a week after the onset. Moreover, we were never able to find any amoebæ. Dysentery, by becoming rarer and rarer coincidently with improved sanitation in this country, and by occurring in only a mild type, has to some extent become unrecognisable. Occasionally, cases are admitted to the asylums. We do not know of any independent inquiry into the conditions of other public institutions, such as prisons or workhouses, but there are certain conditions in asylums which would allow one to believe that dysentery, which in the past affected large numbers of the population of this country, still lurks in asylums, cropping up as sporadic cases, but sometimes endemic in these institutions, and not unfrequently breaking out in epidemic form. Gemmel, in his valuable work, based upon experience in the Lancaster County Asylum, states:—"Dysentery appeared in the reports issued by some asylums, but now the term Colitis, or Ulcerative Colitis

almost entirely supplants it;" he says that the disappearance of dysentery from an asylum where it was previously common, and the substitution of another disease, so identical in its symptoms and post-mortem appearances, and especially fatal in its results, will be difficult of explanation. It may be the term "colitis" being of a non-committal nature, and not connoting defective sanitation in the same way as dysentery, is used because it is less disturbing to the conscience. Gemmel also states that the disease is in great measure limited to larger institutions, and may sometimes account for 33 per cent. of the total deaths.

Griesinger, in his classical work on mental diseases, 1867, states that "Dysentery is very common; epidemics of it have been occasionally observed. The mortality is generally considerable." Griesinger was a distinguished physician before he became an alienist, and he pointed out numerous fallacies introduced into the theory of insanity by the narrow views which had been held regarding it. Griesinger remarks: "Thus, if in an insane person, disease of the spleen or cirrhosis of the liver was discovered, the conclusion was at once arrived at, that diseases of this kind were only to be considered as physical conditions of insanity; examples were collected on all sides, and a theory of the psychical significance of the viscera was framed. Such theories, every day refuted by observation, and at the present time generally abandoned, still occasionally give rise to misconceptions."

The Disease Not Limited to the Insane.—Although the disease is, for the most part, confined to the patients, we were able to collect quite a number of cases amongst the attendants, nurses, and workers living outside, and occasionally even the higher officials have been subject to severe attacks. There are two asylum doctors invited to be present to-night who have suffered with the disease.

Not long ago, my attention was called to an outbreak at a private institution, in which the nurse died. A post-mortem examination was made. Sixteen cases occurred among the inmates, all of which, I believe, recovered. I have little doubt, from personal observations made at the time, that the outbreak started in a chronic case, and spread by contact in the same way as I have seen it do in the large asylums. Also, a considerable number of other cases have been reported in attendants and nurses since the report was issued.

We were informed by two practitioners at Woodford

that married attendants and workmen at Claybury occasionally came to them, suffering with dysentery and severe diarrhœa. Dr. George Turner, in his report on the outbreak of diarrhœa and dysentery in the Melton Asylum, 1894, speaks of fifty-nine cases among females, of which five were among the nurses; twenty-eight male cases, of which one was an attendant.

It is necessary to re-affirm and emphasize these facts, for it has recently been asserted by Dr. Claye Shaw that attendants and nurses are never affected. The symptoms presented by these sane people in no way differ from those of the patients under their care: except, as a rule, they are not so severe, and they are very rarely followed by the chronic form of the disease or by fatal results; but it must be remembered that they are capable of being better nursed, and are better nursed, and have a greater vital reaction.

It is very necessary to consider, as in typhoid, the atypical cases as being forms of one and the same disease; and my experience would show that there are at least seven clinical types. The practical importance of this is shown by the fact that a mild atypical case can be the starting-point of an epidemic in a particular ward. The disease having once started, its virulence seems to increase; even young and physically healthy patients, as well as the old and infirm, are then stricken down; and cases occur of a very severe type, similar to those which affected the prisoners at Millbank, and resembling in onset and clinical symptoms a very severe acute specific fever.

1. The acute case, with preliminary fever, lasting till death supervenes, in two or three days, or within a week.

2. The acute case, with preliminary fever, and a temperature which falls rapidly as the collapse proceeds.

3. The case with mild fever, 101 deg. to 103 deg., and diarrhœa for a day or two, accompanied by diarrhœa with blood and slime in the stools for a few days; terminating, however, in recovery.

4. The mild case without fever, but with diarrhœa, accompanied with blood and slime, lasting over two days. In some of these cases there may have been initial fever, which was overlooked.

5. Cases of varying degree of severity in which, after an interval of a few days, symptoms recur, sometimes with fatal results and sometimes with recovery.

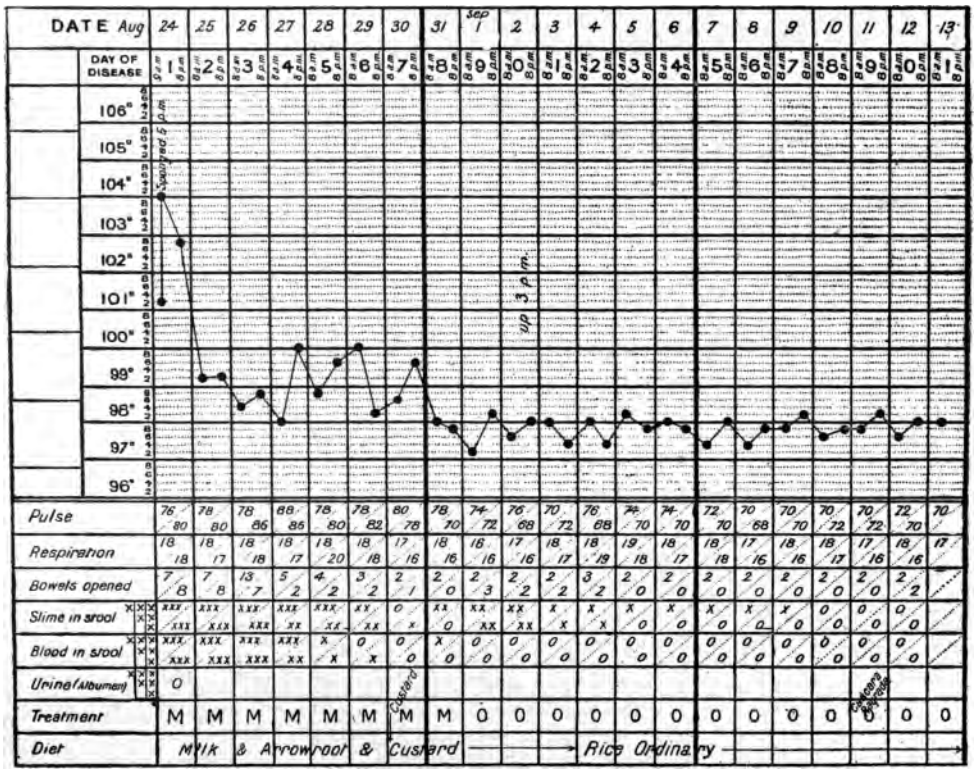
6. Cases which do not clear up after the first week or two, but which become chronic: the patients continuing, at

more or less intermittent intervals, to pass bloody, slimy, diarrhœal evacuations for months.

7. Cases of intermittent or prolonged diarrhœa in which neither blood nor slime has been noticed in the stools, and yet post mortem dysenteric lesions of a similar nature have been found. Some of these (six or seven) are no doubt old cases, which have suffered at some previous period with dysentery. I have numerous records of patients who have had three or four attacks, and at the last succumb. In fact, the majority of the cases which now occur in the returns at Claybury are patients who have had previous attacks of the disease.

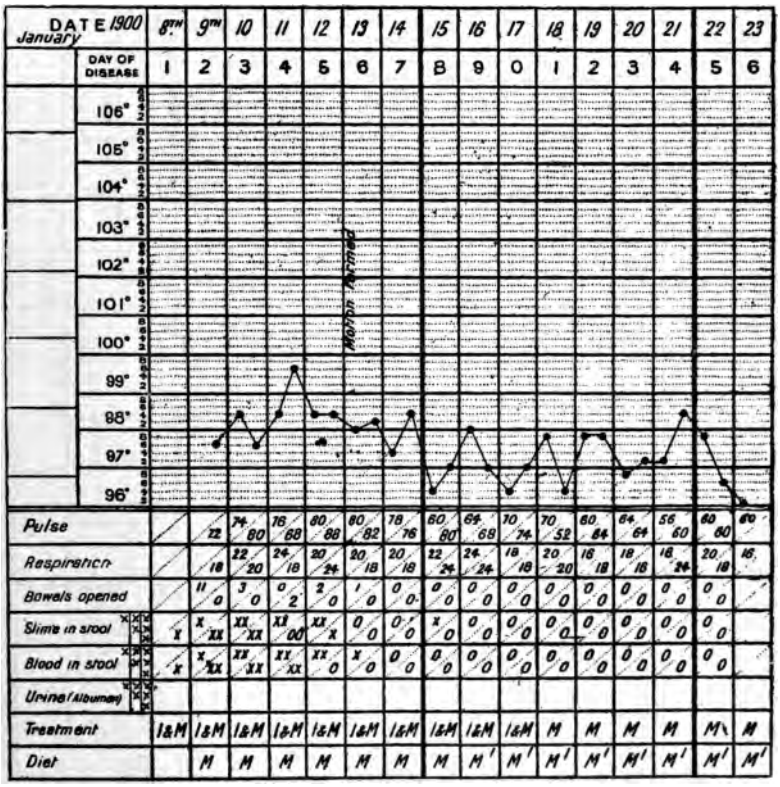
Clinical Phenomena.—Chills or a rigor, accompanied by headache and frequent vomiting, sometimes of a bilious character, associated with high fever, indicate the onset. If the patient be an epileptic or a general paralytic, he may have a fit, or series of fits, and die; the patient may then, if a post-mortem examination is not made, be certified as dying from the fits, or pneumonia, which it resembles so much in its onset. Frequently, indeed, at the post-mortem, associated with the greatly congested red and swollen appearance of the internal lining of the large intestine, there is engorgement of the lungs, especially at the bases, and even pneumonia. In some few cases, the blood-poisoning may be so virulent that there has not been time for the characteristic dysenteric stools to have been observed; and only the fact of an epidemic of dysentery at the time would lead the clinician to suspect the true nature of the disease. The fever, in some of the cases, remains high until the fatal termination; in others, collapse and even subnormal temperature soon supervene. Diarrhœa may come on shortly after the onset, or only after a day or two, soon followed by blood and slime in the stools. Commonly, the disease is of a milder type, and presents the following symptoms:—chills or rigors with pyrexia, followed by frequent evacuations of the bowels, accompanied by pain and tenesmus. Very frequently, however, owing to the mental condition of the patients, pain is not complained of.

Name, Attendant M. O. E.; Age, 26. *Ward. Isolation Hospital, August 24th,* 1899.



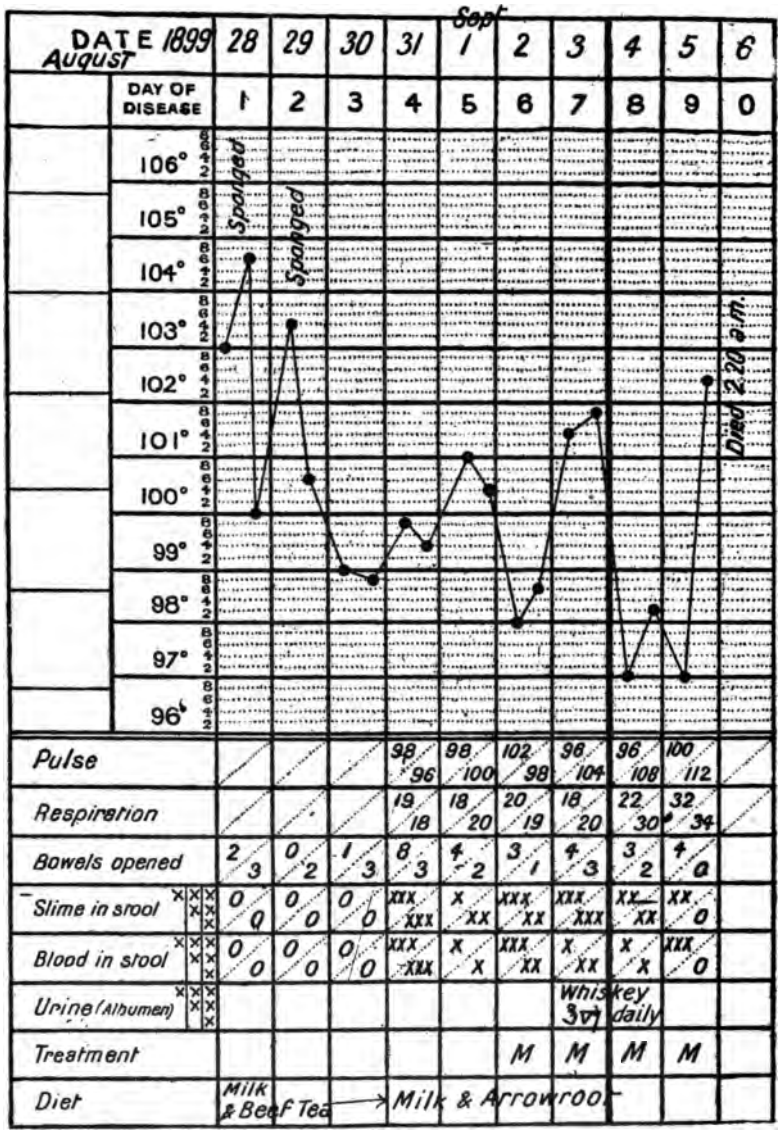
Example of a severe case of Dysentery in an attendant. He was at work during the day in Ward No. 1, Claybury Asylum, and slept in a bedroom off Ward No. 2. It is probable that the patient acquired the complaint in Ward No. 1, where four cases had occurred during the preceding twenty days. The attack began with high fever lasting only one day, and not again exceeding 100 degs. Bowels were moved from fifteen to twenty times a day at first, afterwards less frequently. Much blood and slime were passed during the first eight to ten days, and the slime continued for nearly three weeks, when the patient was pronounced recovered. He is now in good health, and tells me that there is no after-effects of the disease. He was a young, strong man, and no doubt had a high resisting power.

Name, E. A.; Age, 43. *Ward, Hospital.*



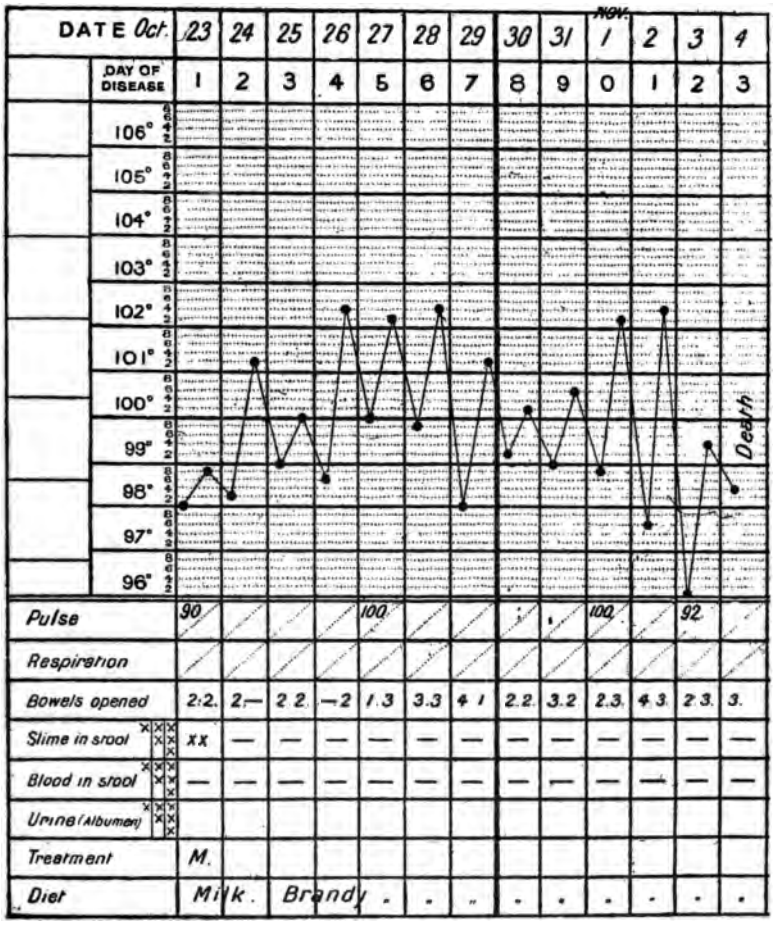
Example of a moderately severe case of Dysentery, with practically no pyrexia, occurring in a woman, the subject of chronic recurrent melancholia. The only diagnostic symptoms of the illness were much blood and slime in the stools. She was admitted to Claybury Asylum, September 19th, 1899, and the dysentery commenced January 8th, 1900. She was treated with Mist. Bism., and Quinine injections. Diet—milk and arrowroot. Recovered on January 23rd.

Name, C. B.; Age, 45. Ward, M. I., August 28th, 1899.
Isolation Hospital, August 31st, 1899.



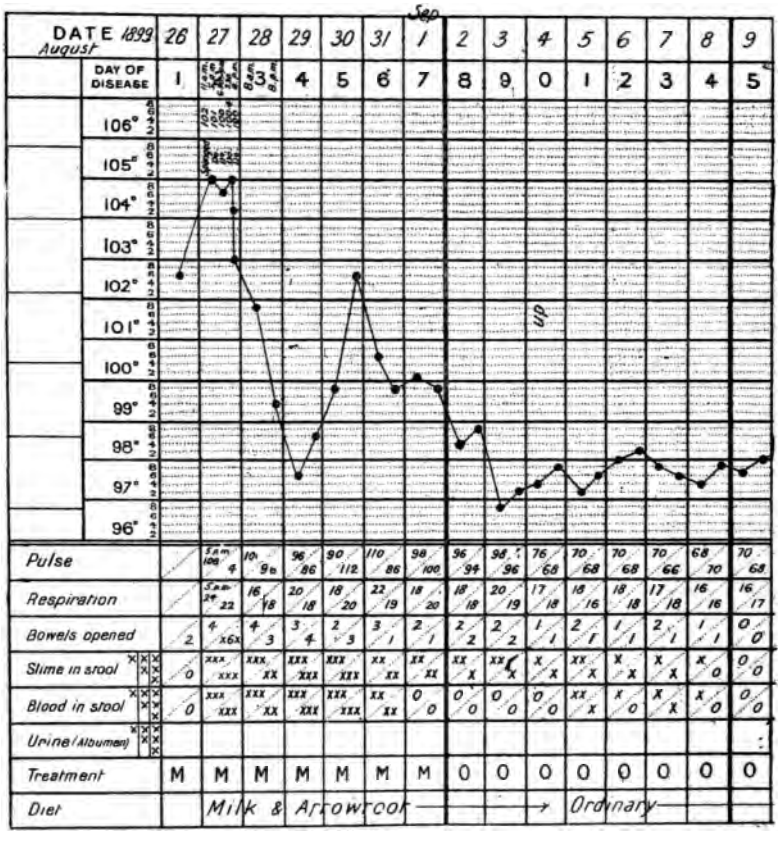
Example of a case of severe Dysentery, which terminated fatally on the ninth day, in a chronic male epileptic, admitted to Claybury Asylum, March 26th, 1895. Seized with slight diarrhœa and high fever on August 28th, 1899. Three days later, when the temperature had fallen nearly to normal, blood and mucus occurred in the stools, and the patient died six days later—the disease being complicated two days before death by broncho-pneumonia. At the autopsy, the whole of the mucous membrane of the large bowel was found swollen, red and inflamed ; there were only a few small ulcers.

Name, C. B.; Age, 38. *Ward,* 2 *I.*



Example of a case of Dysentery associated with pulmonary phthisis, altering the character of the temperature chart. Patient was admitted to Hanwell Asylum, suffering with acute mania, August 18th, 1899, and was attacked with Dysentery October 23rd. The chief symptoms were continual diarrhœa, some mucus at the onset, none afterwards; no blood. Patient was treated with salol, bismuth, and opium mixture; but the case terminated fatally on the thirteenth day. *Post-mortem* examination showed the ascending colon studded with small ulcers, which were discreet with no surrounding inflammation; likewise the transverse colon and descending colon. The sigmoid flexure, the rectum, and the cæcum to a less extent were honeycombed with ulceration; the whole of the mucous membrane was being destroyed, the sub-mucous tissues greatly thickened and of a deep claret colour—the small intestine was free from ulceration. The mesenteric glands were, however, tubercular. Spleen small, pale and shrivelled. The upper lobes of both lungs riddled with cavities. It was extraordinary with such a large amount of ulceration in the intestines that there was no blood in the stools. The absence of ulceration in the small intestine shows that the diarrhœa was not due to tubercular disease of the bowel.

Name, J. G.; Age, 38. *Ward, Isolation Hospital.*



Example of a moderately severe case of Dysentery, occurring in a chronic epileptic employed in the upholsterer's shop, Claybury Asylum, in picking hair of old mattresses requiring to be re-made. Typical sudden onset with shivers, vomiting and high fever, with blood and slime in the stools; treated with mist. bism. c. opio, milk and arrowroot diet; terminating in recovery.

Physical Examination of the patient may reveal some distension of the abdomen, due to moderate tympanites; rigidity of the abdominal muscles, and tenderness on pressure over the cœcum or colon, in its whole tract, or in part. The face is drawn, the tongue red or furred, the pulse frequent and small. The stools may contain much blood and slime, which is of a gelatinous, translucent or opaque, whitish or blood-stained, or greenish (due to altered blood) appearance. Blood may be only recognisable microscopically; and the slime when similarly examined is very frequently found to consist almost entirely of polymorphonuclear leucocytes, decaying epithelial cells occasionally, and a variable amount of mucin. As a rule, however, this is relatively small in quantity. The stools are generally peculiarly offensive, and I can usually smell out a bad case of dysentery in a ward. The attendants tell me they can diagnose a case by the smell of the stools.

I will show a photomicrograph of the slime, consisting almost entirely of polynuclear leucocytes, the cell, *par excellence*, of the blood and acute inflammation. We have found sometimes in cases of more than a few days' duration specimens of the large mononuclear macrophage cell; also, in sections of the colon in similar cases which come to post-mortem table, these and other large cells may be a striking feature of the inflamed tissue. As regards other constituents in the acute cases, passing only blood and slime, one notes a remarkable scarcity of bacteria present. Osler also notes this: when fœcal matter is present, of course, bacteria of many sorts teem in the preparation. If dysentery is caused by any of the "bacilli coli," which have been described by many different authors, their share in the pathology of the disease is very different from that played by the "cholera vibrio" in cholera; for in that case enormous numbers of the vibrios are present, often, indeed, almost in pure culture.

Dr. Durham, who investigated this matter, found that such bacteria as are to be seen in such clear dysenteric stools are of various sorts, ordinary cocci and bacilli of various sizes, some of which retain the stain after iodine treatment, and some of which do not. Besides the above, he observed minute micro-cocci, which he had reason to believe had a possible casual relationship to dysentery.

A negative observation as regards blood and slime by the naked eye does not contravene the possibility of a given case being dysenteric; a case of dysentery does not

always, and may never, pass one of the so-called stools during his illness. This holds not only with the cases we have observed, but also with the tropical disease acquired in the Tropics. Dr. Durham tried the serum reactions in these cases, but without result; and although the leucocytes are so abundant in the stools, we cannot assert that there is any marked increase of the same in the blood.

I shall now pass on to a description of the post-mortem appearance of the intestines, and I may say that, with the exception of a few very acute cases, I have never seen two alike. If the patient succumbs in a very few days after the onset, one finds the walls of the whole large intestine, and frequently the lower part of the small intestine, greatly thickened; the internal lining is swollen and red; often small red hæmorrhagic points or elevations are seen scattered over its surface; the solitary glands, however, are not perceptibly swollen; owing to the marked thickening and swelling of the submucous coats, the mucous lining cannot be moved with the finger over the subjacent tissue.

Microscopical examination reveals acute inflammation of the submucous coat, especially, with engorged vessels and profuse infiltration, which extends into the mucous membrane; and already necrotic changes can be seen to have affected the epithelium, the cells of which are swollen, opaque, bursting, or disintegrated. Photo-micrographs of microscopical sections from such an acute case will be shown.

Acute fatal cases of a little longer duration show the same swelling; but now frequently the mucous lining, although swollen, presents a pale grey or dirty whitish-grey appearance; the surface is sometimes finely or coarsely granular. This is owing to stasis in the vessels of the submucosa and necrosis of the epithelium, and the formation of *a false membrane*, consisting for the most part of leucocytes and disintegrating epithelial cells. A photograph of the bowel in such a case is here shown. The commonest form of the disease met with on the post-mortem table is where some portion or the whole of the large intestine is the seat of ulcers of various sizes, from a pin-point upwards. The wall of the intestine may be thick in one place, thin in another; the seat of necrosis of epithelium in one portion, of ulceration in another; while in other places a healing process has left polypoid elevations or patches of thickened mucous lining. These have rounded edges which are adjacent to portions of the bowel denuded

of mucous membrane; often the submucous coat is destroyed entirely, exposing the muscle, and even this latter may be occasionally partly or completely destroyed. I have seen a few instances of perforation arise from ulceration of all the coats, death occurring from septic peritonitis.

The ulcers are circular or irregularly coalescent; the lumen of the bowel may be greatly increased (as shown in the accompanying photograph), or it may be contracted: as in another case, in which the patient succumbed to an acute attack following three previous attacks, spread over some years. In some cases, in which death occurred from other causes after the dysenteric symptoms had declined, one found pale, somewhat smooth patches, rather more opaque than the surrounding mucous membrane, often more or less invaded by irregular patches of a pinkish or dull purplish hue; these were probably the scars of previous ulceration. Microscopically, they do not show much beyond the wasting of the mucous follicles and increase of interstitial cells. The solitary follicles are frequently seen in these cases surrounded by pigment or a zone of redness.

Post-mortem appearances of the gut may then be most varied; and I see no difference between the appearances presented on the post-mortem table of this Dysentery in asylums to the dysentery occurring elsewhere. It must, however, be clearly understood that, as in the sane so in the insane, inflammatory lesions of the colon or other parts of the intestine, non-dysenteric and non-infective in nature may occur, and that I do not deny the existence of inflammatory lesions of the large bowel, occasioned by Bright's disease, syphilis, etc.; nor do I contest the possibility of stercorous ulcers; but what I do affirm is that these are relatively infrequent and unimportant compared to those of dysentery.

Seeing that, amongst the insane, there is great difficulty in separating clinically such cases from the atypical recurrent or mild dysenteric cases, it is essential that all cases of diarrhœa lasting more than a few days (especially in asylums where dysentery is endemic or has been epidemic), should be regarded as suspect. Again, it is not safe to assume that a patient who has well-marked pulmonary tuberculosis may not also be suffering with dysentery. When blood appears in the stools, or even diarrhœa occurs, it may be due to a dysenteric lesion. I have shown the temperature chart of such a case. Occasionally at Clay-

bury, in recent times, we have had simple cases of tubercular disease isolated for dysentery. Not infrequently one finds old dysenteric lesions of the large bowel with recent tubercular ulceration of the small intestine.

The Hypothesis that Ulcerative Colitis is due to a Nerve Lesion is considered.

Dr. Claye Shaw, in St. Bartholomew's Hospital *Reports*, 16th vol., 1880, p. 9, "On some Intestinal Lesions in the Insane," has asserted that ulcerative colitis is a common termination in chronic dementia and general paralysis, the primary cause being nerve degeneration.

In a quite recent Paper at the pyschological section of the last British Medical Association, he commented on our report, in support of his theory that ulcerative colitis is due to a nerve lesion, thus: "For this assertion one ought to be able to give some cogent reason; unfortunately, I cannot adduce any actual observations to prove that there is a nerve degeneration; but, judging from the analogy of the tissues, it is extremely probable that this is the case. One has only to look at the degeneration of all the tissues in chronic insanity—degeneration from which any organ never seems exempt—to make it appear more than strange if the intestinal system was not affected in the same way; and, if so affected, it would take the form of sloughing ulceration or large patches of obliteration." Dr. Shaw also says: "I quite agree with the recommendation of Drs. Mott and Durham for isolation of dysentery cases;" but he contends that the condition we described was *exceptional*, and that there is a group of asylum cases of colitis or Asylum Dysentery—if you please to use such a term—dependent on "nerve degeneration or toxic causes set up spontaneously in the individual." I am glad to note that we have now his permission to use the term "dysentery;" but for practical purposes I fail to see the value of his arguments, for, further on he states: "I confess, however, that the symptoms in the two forms are similar, and that a distinction is to be made at times only by a very exhaustive analysis of the conditions." We should therefore either be bound, according to his own showing, to isolate all cases, or regard some as suspect, and requiring special precautions if the cases are not segregated from the rest of the patients.

I have seen several instances in which an epidemic of dysentery apparently started from a mild atypical case,

which probably Dr. Shaw would have considered due to nerve degeneration; and I think, from his statement afterwards, Dr. Shaw himself is quite prepared to admit this, for he states: "I contend that all cases of colitis, with or without ulceration, are not primarily bacterial in origin." But now comes the important part of his statement, and this I wish to lay stress upon: "I do not deny that bacteria are secondarily present, and that then the cases may become infectious, if the discharges are not properly treated and the nursing is insufficient." It was precisely those conditions that we found defective in some of the asylums where dysentery was prevalent. Apparently, from the returns made from Banstead and Canehill, epidemics such as we met with at Claybury, Hanwell, Colney Hatch and Bexley, did not prevail. But to return to the possibility that simple cases may become infective, we must assume, in the absence of any precise knowledge of the specific agency which would cause them to be infective, that organisms that find a normal habitat in the intestine may take on, under unfavourable sanitary conditions, a virulent character: somewhat as we may suppose a foul sloughing ulcer introduced into a hospital ward, in olden times before antisepsis and principles of cleanliness were adopted, would sometimes cause spreading gangrene; but the occurrence of this foul disease was condemnatory of the sanitary arrangements of the hospitals in which it developed; and I quite agree with Dr. Shaw that it is impossible to prevent sporadic cases of intestinal lesions, which I term dysentery, and attribute in great measure to the present system of filling up new asylums from old asylums with transfers where the disease may have been present. Dr. Shaw would attribute these sporadic cases to nerve degeneration, while admitting that they may serve as a focus of infection if the hygienic conditions of the asylum are defective.

The following facts are against Dr. Shaw's views with regard to ulcerative colitis being due to nerve degeneration associated with insanity.

1. There is no essential difference between the lesions of the chronic ulcerative colitis one sees in the sane, which I should regard as chronic dysentery, and the sporadic cases of ulcerative colitis met with in asylums.

2. If this condition were due to a nerve lesion, there should be a proportional relationship of sporadic cases among the insane of all asylums, which is not the case. According to my experience, these sporadic cases are much

more numerous in asylums where there there has been previously an epidemic. Claybury Hall, which is the old mansion of the estate, holds fifty-eight patients; as far as I know, and I have reason to believe the statement is true, there has not been a single case of dysentery, acute or chronic, for six years, since it has been used for patients. It might be argued that the healthier and less demented patients only have been lodged there. For the past two years, however, this building has contained only private patients, and 25 per cent. of these patients have been general paralytics. This is of importance as disposing of the theory that intestinal lesions occur with greater frequence in general paralysis; it also disposes of the idea that the soil, the food, or the water, which is the same as in the large building, was the cause of the disease. Examination of the splanchnic nerves of the most severe case of chronic dysentery I have ever seen, photographs of which I have shown you, revealed no degeneration. It may be presumed that the ulceration in this case was not associated with primary degeneration of nerves. I have also examined the sympathetic nerves in the mesentry, in a case of chronic insanity, by Dogiel's Methylene blue method, without discovering any recognisable change, although this patient had suffered with chronic dysentery. Again, by the chloride of gold method, I have examined the plexuses of Meissner and Auerbach in several cases of early general paralysis, without discovering any change whatever. I do not, however, claim for these examinations any more than this: that they are of some value, whereas an hypothesis like Dr. Shaw's, based solely on analogy, is valueless, unless some more striking evidence can be brought forward in support of it.

3. One would expect intestinal lesions to occur in the tabetic form of general paralysis if this theory were correct. I have recently completed the examination of a case of this kind, in which all the dorsal and lumbo-sacral posterior roots were in a condition of advanced atrophic degeneration, the visceral fibres were therefore also markedly degenerated, and yet the internal lining of the whole intestine was normal.

4. My experience coincides with that of Griesinger: that neither acute nor chronic dysentery appears to have any permanent effect upon the mental symptoms of the insane.

Etiology.—The question of the existence of a specific organism is of great importance. Dr. Durham did not attach

much importance to the colon-like bacillus of various authorities: Shiga (Japan), Kruse (Germany), Celli (Italy), and which has been called the "bacillus dysenteriœ." They were unable to reproduce the disease in animals; but recent work seems to show that although inoculation with this bacillus does not produce dysenteric lesions of a characteristic nature, yet it produces a change in the blood of animals similar to that in man suffering with dysentery, as regards agglutinating reaction of the serum on this motile bacillus. Shiga was the first to demonstrate this; and recently an important communication of Flexner has appeared in the *Centralblatt für Bakteriologie*, October 12. 1901. He refers to the observations of Kruse *F. N.* (*Deutsch, Med. Wochenschrift*, 1900, p. 40), who published an account of the bacteriological examination of an epidemic of dysentery, which occurred in the previous July, in the manufacturing town of Laar in Germany. The epidemic affected three hundred persons, and caused thirty deaths. Kruse isolated a bacillus which he regarded as the cause of the disease. It closely resembled B. Typhosus, and the agglutination reaction with the blood of dysenteric patients was unmistakeable. Kruse regarded this organism as related to, but probably distinct from, the bacillus of Shiga. Dr. Durham found no agglutinating reaction of the blood of dysenteric patients at the dilution of 1·100 with B. Typhosus, or B. Enteritidis. He did not, however, use the B. Dysenteriæ. Kruse *F. N.* (*Weitere Untersuchungen*, etc.) has added a paragraph on the "pseudo dysenteries of asylums." While it is admitted that epidemic dysentery may be introduced into asylums, the dysenteries so commonly found among the insane are regarded as distinct, and probably of different and variable origin. Whether closer study will confirm these views remains to be seen. Kruse examined material from twelve stools and two autopsies, and comes to the following conclusion:—"I was not able to find in any case bacteria possessing all the properties of the dysenteric bacillus."

On the other hand, Flexner obtained from one stool and from both autopsies a bacillus whose morphological and cultural properties could not be distinguished from those of B. Dysenteriæ. The only point of difference was found in the reaction of the specific dysenteric blood-serum. The blood of the asylum cases (11 out of 15) agglutinated the three strains of bacilli in dilutions of 1 : 100, or more. But the blood had no effect upon the true dysenteric bacillus. "Kruse concludes that in asylums a form of

dysentery occurs that has nothing to do with true dysentery, but which probably owes its origin to similar micro-organisms of which there are probably several distinct types." Flexner's observations, however, shew that the B. Dysenteriæ is the same for all countries and asylums. He regards Kruse's conclusions as premature, and he points out that two cultures of Philippine dysentery bacillus which he sent to Kruse were found by him to agree culturally with those he (Kruse) had obtained from asylum cases.

Again, Flexner has studied two cases of dysentery which developed in the wards for the insane at Philadelphia Hospital, and the blood of these patients gave positive results. If now there is a bacillus which is contained in dysenteric stools found in all forms of dysentery, as Flexner maintains, we may conclude there is a specific organism of dysentery which is the cause of the infection, and the serum reaction might become a valuable test, and a means of helping in the diagnosis of the atypical suspect cases. I have written to Dr. Flexner, asking him to kindly send me a culture of his bacillus dysenteriæ, in order that I may try the agglutinating reaction of serum obtained from patients suffering with the disease in the London County asylums. I should think it quite possible that some recurrent cases would not give the reaction. If Flexner's observations are substantiated, it would show the unity of different forms of dysentery in different countries.

The consideration of the occurrence of dysentery in particular wards of asylums leads to the conclusion that it is communicated by infected patients to those previously free, in some direct or indirect manner. The possible modes of communication are due in part to the ignorance of the attendants, who have not appreciated the communicability of this disease, or exercised sufficient care in preventing its spread by attending to their own personal cleanliness, or the personal cleanliness of the patients, *e.g.*, in the matter of washing hands after assisting in menial ward duties; which include, I may remind you, cleaning up patients, taking away foul linen, and the handling of utensils and food in the preparation for meals.

Some instances of outbreaks attributed to these causes I will now bring forward. In a new temporary ward at Colney Hatch, the diagram of which will be found on p. 42, an outbreak occurred: ten patients were affected, eight of them occupied adjacent beds, and were successively

affected; seven of the ten died. Most of them were old and bedridden, and it is presumed that the attendant may have been the means of communicating the disease.

An outbreak which I investigated at a private asylum began in an atypical case, spread to patients in three adjacent single rooms; the night nurse became affected, as also a number of cases whom she attended at night. The nurse died, and a post-mortem was made, with the result that an acute dysenteric inflammatory affection of the large intestine and lower part of the small intestine was found. The fact that only the inmates of the asylum who were under the care of this nurse were affected, appeared to me to exclude the food, milk, and water supply.

Outbreak of Dysentery in Ward 20, Male Side, Hanwell.

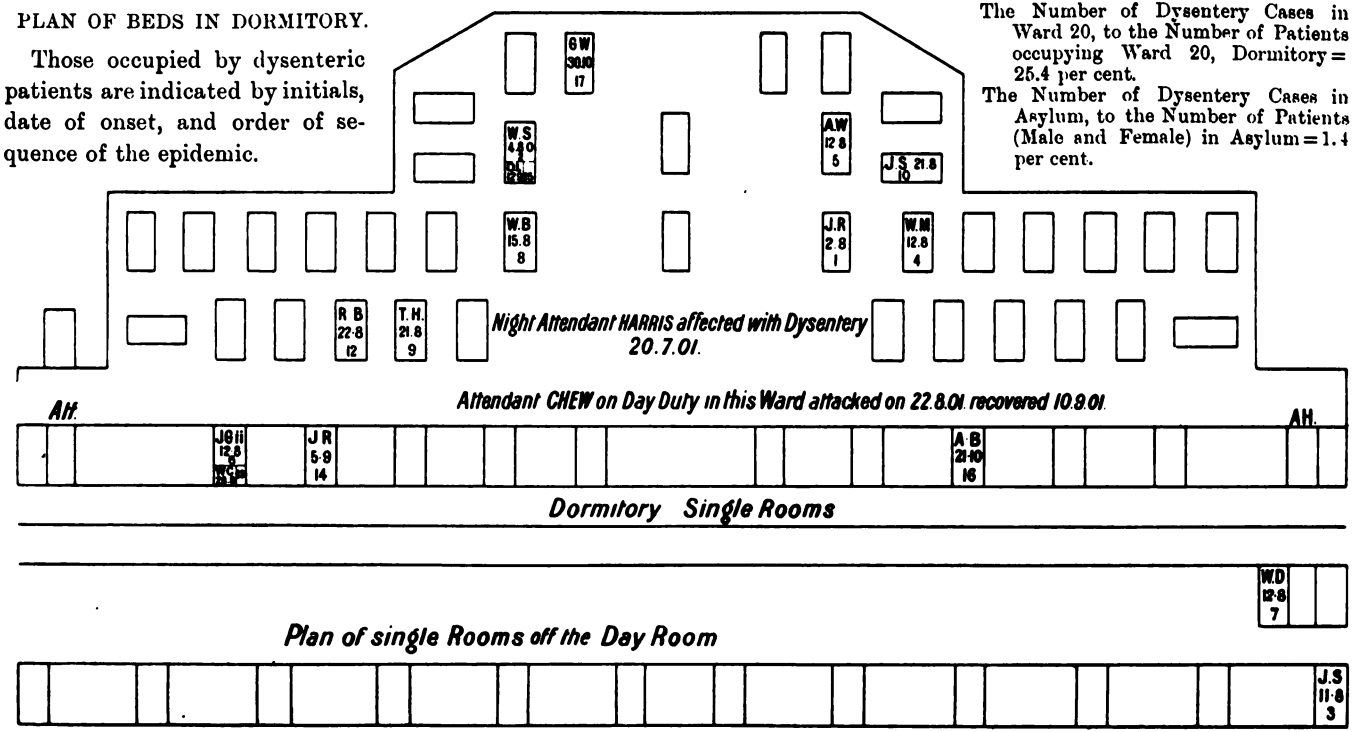
All cases of dysentery and diarrhœa had been notified since March 2nd and not a single case had been returned from this ward. In August, twelve cases of dysentery and several of diarrhœa were reported from this ward. The case first notified was in a night attendant. On the night of Saturday, July 20th, the night attendant Harris, a man who had been thirteen years in the service, suffered with slight colic and nausea, but no diarrhœa. He went home, and the pains became much worse. At noon on Sunday he passed the first loose motion, another at 1 p.m., after which diarrhœa became severe, and the third motion contained a little blood. He continued to pass blood and slime throughout Sunday and Monday. He was seen by Dr. Worthington, who certified him suffering from enteritis; but, finding his treatment did not do him any good, on Tuesday night he took castor oil, and felt greatly relieved the next day. On Wednesday, July 24th, the diarrhœa had entirely stopped; and he felt perfectly well. He remained off duty until the following Tuesday, July 30th, and had no recurrence of symptoms during the interval. He is unable to recall any definite case in the ward prior to his own, but is under the impression that there has been one. A reference to his own night-book, however, fails to record any case, and none can be traced. I am indebted to Dr. Spark for the above information.

HANWELL ASYLUM, WARD 20: MALE.

From August 1st, 1901.

PLAN OF BEDS IN DORMITORY.

Those occupied by dysenteric patients are indicated by initials, date of onset, and order of sequence of the epidemic.



The Number of Dysentery Cases in Ward 20, to the Number of Patients occupying Ward 20, Dormitory = 25.4 per cent.

The Number of Dysentery Cases in Asylum, to the Number of Patients (Male and Female) in Asylum = 1.4 per cent.

I visited Hanwell Asylum on December 5th and saw Dr. Alexander, who stated that Ward 20 was a new building; the drains had been in perfect order, and he could not account for the outbreak, unless it was due to a drain being opened in the airing court in which the patients of this ward, together with the patients of Wards 1 and 2, took exercise. The fact that no cases occurred in these latter patients, only in those of Ward 20, negatives this supposition. At Dr. Alexander's request, I visited Ward 20; and I should here again like to take the opportunity of speaking to the courtesy and readiness with which I have always obtained information from Dr. Alexander and all the members of his staff, and their desire to do all they can to assist me in investigating the cause of the disease. In the company of Dr. Spark I went to Ward 20, and saw first Second Charge Attendant Thomas Farrance. He stated that there had always been one or two cases in this ward, even before Attendant Harris had been affected [As they were not notified, they were probably mild cases.—F.W.M.]; and it is quite likely that he should have taken the disease from one of them. In the early part of August, a lot of cases of dysentery cropped up in the ward, and Attendant Chew, on day duty, was attacked on the 21st of August.

Attendant Chew, aged 25, was in the asylum service six months before he suffered with the disease. He had served previously in a line regiment, and had been in India. In answer to a question, he stated that he had not been told that the disease was infective. His duty was to see to the patients, wash, and change them. He stated that he had seen plenty of dysentery in India, while in the army; and in answer to a question, he stated that the only difference he found was, that this disease is worse and more serious: more slime and less blood. He made the following statement with regard to his own attack: August 21st, he was ordered by the Charge Attendant, Mr. Whitely, to cleanse one of the patients named Osborne, and the bed was in such a frightful state with blood and slime, he felt upset. At night he started vomiting, and reported himself to Dr. Thomas, and was sent to bed. He felt very feverish, and had a bad headache, with loss of appetite. On the second day he commenced to pass blood and slime, and was on the stool nearly all night long, and thought he was going to die. Dr. Thomas said he was one of the worst cases he had seen. He continued to pass blood and slime for ten days or more. The fever continued for a week, and he was very thirsty.

For two or three days after the onset he suffered with attacks of vomiting, and for a fortnight he had pains in the belly, gripes, and straining at stool. He was very tender over the belly during this time. He was off duty about one month. He never had it in India, although he had nursed a number of soldiers with it, a few of whom had died. He considers he had as severe an attack as the soldiers he saw in the hospital in India.

It is quite true that while changing a dysenteric patient, the attendant may have to rush off to restrain a patient who is getting out of bed, or creating a disturbance in the ward. Many times he has to rush off from changing a patient, to attend to other ward duties, without having time to cleanse his hands. He thought the handling of eating utensils and of food, under such conditions, might easily be the means of communicating the disease. Since the outbreak, feeding-cups, knives and forks of each patient are kept separate. The ward is well ventilated; and, except the first thing in the morning, there is no offensive odour, except when there are dysentery cases, and then the smell is very foul and characteristic: stronger here than the smell in India.

The most probable origin of the epidemic in this ward was the existence of several cases of mild atypical dysentery, which had not been notified, but which there is reason to believe, from the statement of Attendant Farrance, existed. The existence of such cases in the ward was probably the cause of infection of the night attendant, who may, as well as getting it himself, have been the means of conveying it from one patient to another. The statement of Attendant Chew shows the absolute necessity of isolating in a separate building cases of dysentery; or, if that be impossible, of employing an attendant skilled in nursing communicable diseases, who should have no other duties than looking after these patients.

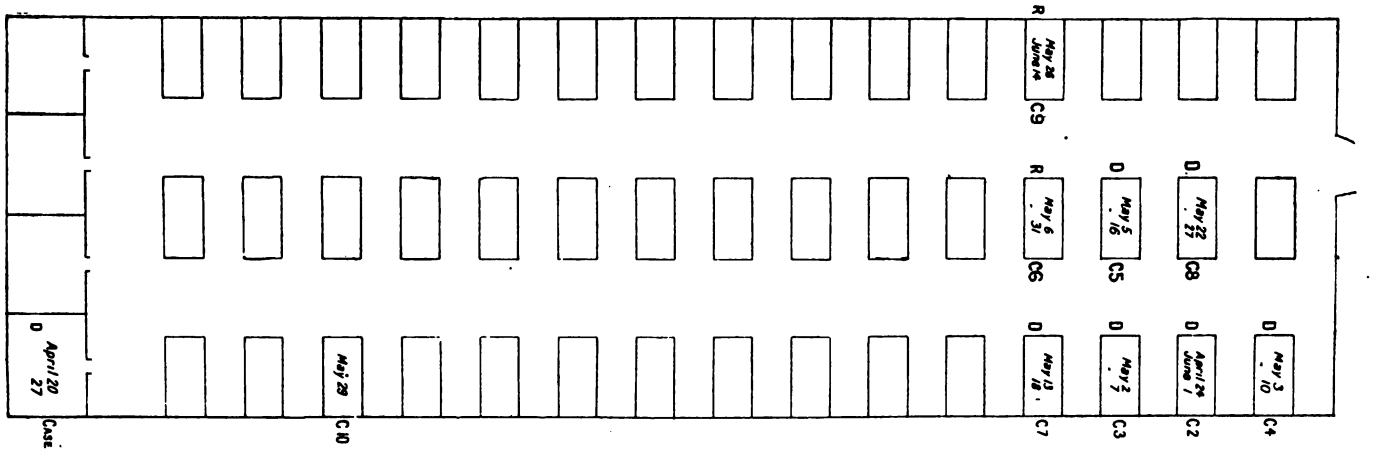
Fifteen patients suffered with dysentery and four with diarrhœa in this ward; 35.5 per cent of the inmates of this ward, and 57.5 per cent. of the dysentery cases of the whole of the male patients. The total percentage of dysentery for the whole of the inhabitants of the asylum was 1.4 per cent. only. The dysentery cases in this ward, 20, made up 40.5 per cent. of the total dysentery cases of the whole asylum. It thus shows that much asylum dysentery is due to communication of the disease from one patient to another, probably by ignorance, carelessness, or deficient precaution on the part of attendants. For, if

attendants can acquire they can confer the disease; and it emphatically shows the necessity of isolation of infective cases, and nursing by attendants who have been properly instructed in the nursing of typhoid cases. The same precautions require to be taken for dysentery as typhoid. We found that typhoid, which has been recognised by asylum authorities as liable to be conveyed from one patient to another, still, with the precautions that are taken at present, frequently spreads. It is seldom that a case of typhoid remains solitary; usually a crop of cases occur. This is at present the case at Colney Hatch, where dysentery is also prevalent.

In many respects, the sanitary condition and hygiene of asylums where dysentery was prevalent were not good: *e.g.*, at Claybury, the drains were all defective wherever tested, and leaked into the subways that served to supply, in the winter time, the warm air to the building. During the prevalence of the disease at Claybury, we found lettuce-beds irrigated with raw untreated sewage. At other asylums we found imperfect destruction or sterilisation of articles known to be contaminated. In one asylum, foul mackintosh sheets were being washed in a bath; patients washed their hands at slop sinks; and, during our presence in the ward, the excrement of a known case of dysentery was disposed of down the same sink; within a few yards was the proper, but locked, ward lavatory. The care in personal cleanliness on the part of the attendants and nurses in the wards where dysentery was prevalent required supervision and improvement; the washing of hands of patients in the lavatory, at any rate, only took place in the morning, when they got up, the lavatory door being locked, towels and soap being all ready for the next morning. I was glad to find, on a visit to some of the wards at Claybury, a short time ago, the patients using the lavatory to wash their hands before having their meals; and to this attention to personal cleanliness, as well as to the greater care exercised by the attendants in the recognition of the disease, the knowledge of its communicability, and immediate reporting of the case to the medical officers, together with the diligence with which the medical officers themselves have endeavoured to practise early isolation, I attribute in great measure the steady decline in the number of cases which have occurred, especially on the male side, at Claybury since our recommendations have been adopted.

The segregation and disinfection of things which come

Plan of X 1 Temporary Buildings, Colney Hatch, in which an Outbreak of Dysentery occurred in April, 1900, *which shows clearly communicability of this disease.*—F. W. M.



The date of onset and duration of the disease in each case is indicated in the space representing the position of the bed; those with D indicate that there was a fatal termination; those with R indicate recovery. The order of sequence of onset is indicated by C and a numeral.

into contact with patients afflicted with any form of diarrhœa required more stringency and method, *e.g.*, the use of disinfectors, but especially the cleaning of mattresses, and the removal of foul linen. Mattresses at Colney Hatch and Hanwell were most imperfectly dealt with; in fact, we found experimentally, that the hair of the mattresses which were supposed to have been cleaned, contained a larger number of bacteria than before. Another point was the absence of any distinguishing mark, so that contaminated mattresses from one ward "thus cleaned," could be returned to another ward, and, if infective, start disease there. This laxity in sanitary practices suggested defective supervision in these vast establishments. The desirability of keeping systematic notifications of all diarrhœa cases and systematic records of suspicious cases, is obvious from the fact that it is not improbable that the continued presence of dysentery in asylums may be in great part due to the lighting-up of old cases, and the neglect of precautions in mild and transient cases. It is only fair, however, to point out the great difficulties attendant on the discovery of all these cases among the insane, who are generally mentally incapable of telling you their complaint, and are naturally dirty in their habits.

Isolation Hospitals.—Although buildings are provided for isolation they were not available for use, as they were filled up with ordinary chronic lunatics. This was the case at Claybury, Hanwell, and Colney Hatch. The new asylum at Bexley does not apparently possess an isolation hospital. At present, at Claybury, the isolation hospital is filled with female patients who have suffered with a previous attack, or are at present suffering with dysentery; the male patients being isolated in a small separate building. The question of housing the large number of lunatics in the County of London has been a most pressing problem, and the Council have had to consider the ratepayer as well as the pauper lunatic; that there has been great overcrowding, no one will deny. I do not consider overcrowding in itself such an important cause of spreading the disease as the various shifts that have to be resorted to, to make accommodation for patients, such as the filling up of isolation hospitals, and the transference of cases which have suffered with dysentery from one ward to another. Thus, in the Report of the Pathological Laboratory, May 21st, 1901, I pointed out that one of the female patients who had died of chronic dysentery was in twenty-three different wards in five-and-a-half

years, including four periods in the isolation hospital, during the last of which she died. Another was in sixteen wards in seven years, including two periods in the isolation hospital, and eventually died in an infirmary ward directly from dysentery. Another was in fourteen wards in four-and-a-half years, including three periods in the isolation hospital, during the last of which she died. Another was in thirteen wards in fourteen months, including three periods in the isolation hospital, during the last of which she died. Finally, a fifth patient was in thirteen wards in three years, and was never in the isolation hospital at all. Seeing that we have definite evidence, in the case of two laundrymaids at Hanwell, of a recurrent case being infective, we see how important this matter of transfers is; and it also shows the necessity of the system of notification which has been in force, as well as an efficient system for the separation of dysenteric patients from other patients. Moreover, it shows that great care should be exercised in the opening of new asylums, not to admit old cases who have had dysentery.

There is no reason to think that dysentery has spread in the London County Asylums owing to the water-supply. A return of cases of enteric fever which occurred made it probable, from the data supplied, that this fever had been passed from one patient to another in certain cases. The outbreak of dysentery, and the mode of its spread in particular, as I have shown, would suggest that it could also be communicated from patient to patient in this way. The facts of the distribution and the occurrence of the disease are not in accordance with the supposition that it is acquired through the ingestion of unsound food, although it is highly probable that indigestible articles of food, *e.g.*, pork, unripe and unsound fruit, may light up latent dysentery in individuals who have previously suffered. In certain asylums, *e.g.*, Claybury, the drains were defective, and leaked into the subways; but, inasmuch as outbreaks of the disease occurred in the summer and autumn, when this system of ventilation, by air from the subways, was not in use, it was not considered that the dysentery was directly associated with this cause. Moreover, the limitation of the disease to the patients, and the escape of the staff, except those who were in direct attendance on the patients, make a defective drainage system an improbable factor in the spread of the disease. At Rainhill Asylum, where the disease was rife some years ago, Dr. Wigglesworth, the

superintendent, told me that he looked upon the disease as communicable like typhoid, practised isolation, and had a system of destroying all the dysenteric stools in a cremator; by doing so, the disease only occurred in sporadic cases now. I am convinced that the continuance of notification, with relief of overcrowding and its dependent sanitary defects, the treatment of the cases like typhoid, with constant and minute attention to recognised modes of isolation on the part of the staff, attendants, and nurses in asylums where dysentery exists, will much abate the disease; and when cases do arise, either within the asylum, or from transfers or admissions, the spread of the disease will thereby be arrested.

A substantial support of the infective nature of the disease, and of the value of the adoption of the methods that are practised for the isolation of patients suffering with it, has been afforded at Claybury; and I am pleased to show you evidence, obtained in the post-mortem room, indicating a marked diminution of the disease.

In conclusion, I wish to thank the superintendents and officers of the various asylums for the readiness with which they have afforded me information.

Postscript.

Since reading the Paper, interesting and important facts relating to the infective nature of the disease have come under my observation. I am indebted to Dr. Bailey and Dr. Baker for the following notes.

Note by Dr. Bailey, of Hanwell.

The following members of the staff have suffered with the disease at Hanwell:—

Males.

	Duration.
Dr. Bailey, August, 1900	14 days.
Talbot (Store-room Porter), March, 1901 ...	3 weeks.
Attendant Austin, July 18th, 1901 ...	9 days.
„ Muds, July 16th, 1901	3 weeks.
„ Chew, August 22nd, 1901 ...	3 weeks.
„ Burge, October 31st, 1901 ...	5 days.
„ Harris, July, 1901	9 days.

Females.	Duration.	
Miss King, Matron, Summer and Autumn, 1899.	3 months (pensioned).	
Nurse Tibbot, April 6th, 1901	1 week.	
Laundrymaid Spray, April 22nd, 1901 ...	2 weeks.	
Head Attendant Brown, September 23rd, 1901.	3 weeks.	
„ Nurse Weston, October 21st, 1901 .	5 days.	
Laundrymaid Spray, December 13th, 1901 ...	Recurrence	Occupying a two-bedded room.
„ „ Weston, December 16th, 1901 ...	Continuing	

Dr. Bailey has also called my attention to the fact that in two cases, one of dysentery, another of probable dysentery, occurring at Hanwell, a liver abscess was found *post-mortem.*

Note by Dr. Baker, of Hanwell.

Two Cases of Dysentery occurring in two Laundrymaids occupying a Two-bedded Room.

Illustration of a recurrent case infecting a healthy person. The disease the same in its clinical characters as that affecting the insane patients of the asylum.

Case I.

Spray, L.M., Ward 20, sleeps in a two-bedded room, with Brooks, L.M., over the laundry.

Spray, L.M., began to suffer with diarrhœa and abdominal pains on April 16th, 1901. She remained "on duty" for a week, although the diarrhœa did not stop. On April 23rd, she became so ill that she applied for medical treatment. She was then found to be suffering from severe diarrhœa, with both blood and slime motions. She was at once isolated in the "hospital," with several other patients who were suffering from a similar condition. After being in the "hospital" for a fortnight, she returned "on duty," cured.

Temperature chart during treatment in "hospital" is enclosed.

Brooks, L.M., who slept in the same room, did not develop symptoms of colitis.

Spray, L.M., remained free from "colitis" till December 11th, 1901, when diarrhœa and abdominal pain again occurred. There was blood and slime in the motions on December 13th. On the 14th of December she first came under medical observation, and was found to have "recurrent colitis."

During the whole time of her second illness, Spray, L.M., has been sleeping in the same two-bedded room, with Davis, L.M., in which Spray, L.M., developed her first attack.

Spray, L.M., is still under treatment for diarrhœa, with blood and slime in motions, and abdominal pain. Temperature has not been over 99.2 deg. F. at night.

Case II.

Davis, L.M., Ward 20, sleeps in the same two-bedded room, with Spray, L.M.

On Dec. 14, 1901, when Spray, L.M., was found to have "colitis," Davis, L.M., was ordered not to sleep in the same room.

On the night of the 14th, she slept "at home," and on the 15th, in a three-bedded room, with two nurses, in the temporary buildings. On the morning of the 15th she became ill with

vomiting, abdominal pain, and diarrhœa. While "at home" she noticed some blood in the motions.

She first came under medical observation on the 16th of December, and has been isolated with Spray, L.M., in the Nurses' Block.

A. E. BAKER.

Hanwell, December 16th, 1901.

APPENDIX TO PAPER.

Report on Dysentery in Asylums, March to November, 1901 *inclusive.*

CLAYBURY ASYLUM.

Male Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	6	5
April	—	1
May	2	6
June	1	1
July	1	1
August	6	5
September	5	2
October	4	—
November	3	2

The cases do not appear to have occurred especially in any particular ward. No cases of bowel disturbance have been reported from the staff. From April to July the cases of dysentery and diarrhœa were very few in number, but recrudescence of the disease occurred in August: the patients who were affected, however, being those who for the greater part had had one or more previous attacks.

1899.	Dysentery Cases.	Diarrhœa Cases.
August 12th to 31st	15	2
September	18	4
October	6	—
November	5	—

The above figures include 2 cases of dysentery and 2 cases of diarrhœa, which occurred among the staff.

Female Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	9	25
April	3	19
May	4	19
June	2	5
July	2	3
August	15	3
September	9	2
October	9	1
November	4	—

Cases do not appear to have occurred especially in any particular ward.

	Dysentery Cases.	Diarrhœa Cases.
Ward D 1	6	1
Ward H 2	4	15
Ward D 2	4	—

The above figures include 9 cases from the female staff, one of whom suffered with an attack of dysentery. (She was charge nurse of the Isolation Hospital.)

1899.	Dysentery Cases.	Diarrhœa Cases.
August	16	8
September	3	14
October	9	—
November	6	1

These figures include 6 cases of diarrhœa, which occurred among the female attendants.

HANWELL ASYLUM.

Male Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	1	7
April	1	8
May	—	4
June	1	4
July	—	9
August	19	20
September	4	4
October	2	2
November	2	9

During the month of August an outbreak of dysentery occurred, when the number of cases reported was 18, as against *nil* in the preceding month; 18 cases of diarrhœa were also returned. One male attendant suffered with dysentery, and 6 with diarrhœa. The majority of the cases reported in August came from Ward 20, viz., 10 out of 18 cases; 3 cases of diarrhœa also occurred here. The only male attendants reported as suffering from dysentery in all the asylums during the past nine months came from this ward, where so many cases had occurred. During the last three months 4 more cases of dysentery were reported from Ward 20.

1899 (1st to 31st).	Dysentery Cases.	
August	3	No data as to diarrhœa cases.
September	1	
October	2	
November	—	

Female Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	1	15
April	5	38
May	3	7
June	—	2
July	1	4
August	7	17
September	2	5
October	2	5
November	—	4

No particular wards were affected. A comparatively large number of cases in April and August, while very few cases were recorded in June, July, October, and November. One female attendant was returned as suffering with dysentery, and one with diarrhœa, during the nine months.

1899.	Dysentery Cases.	
August 1st to 31st	22	No data as to diarrhœa cases.
September	5	
October	2	
November	1	

HORTON MANOR ASYLUM.

Male Side.

1901.	Diarrhœa Cases.	
March	—	No dysentery cases reported.
April	2	
May	—	
June	1	
July	—	
August	2	
September	—	
October	1	
November	—	

A few cases of diarrhœa have occurred, scattered through the wards on male side. One attendant suffered with diarrhœa.

Female Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	—	—
April	2	2
May	1	—
June	—	—
July	—	—
August	1	1
September	1	2
October	—	1
November	—	—

Attack not confined to any particular ward. No member of the staff has been reported suffering with any bowel disturbance.

CANE HILL ASYLUM.

Male Side.

1901.	Diarrhœa Cases.	
March	—	No dysentery cases reported.
April	1	
May	4	
June	—	
July	—	
August	4	
September	—	
October	1	
November	3	

The male side of Cane Hill Asylum, for the past nine months, has been by far the most free from dysentery and diarrhœa cases, as compared with the numbers from the other asylums. Only 10 cases of diarrhœa have been reported, 3 of these occurring among the male attendants.

Female Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	2	5
April	1	7
May	—	6
June	1	5
July	—	4
August	1	7
September	—	1
October	—	4
November	—	22

During the past nine months 5 cases of dysentery have been reported from Cane Hill Asylum, female side, no particular ward being affected. Ward J2, however, reports 1 case of dysentery and 12 cases of diarrhœa. The monthly statistics include 7 cases of diarrhœa, which occurred among the female staff.

In the latter part of November, a general attack of diarrhœa took place in Ward A1, lasting for two days; 13 patients suffered. The direct cause was not ascertained.

BEXLEY HEATH ASYLUM.

Male Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	2	5
April	3	4
May	7	4
June	2	41
July	1	43
August	1	19
September	—	26
October	—	12
November	—	8

During the past nine months Ward J1 accounts for 22 diarrhœa cases; Ward K1 for 11 dysentery cases and 18 diarrhœa cases. In the month of May, when dysentery was on the increase in this asylum, the whole 7 cases for the month came from Ward K1, and in June the two reported cases for the month were also furnished by this ward. During the nine months 11 of the 16 reported dysentery cases came from Ward K1. Only 1 case of diarrhœa is reported from the male staff. During the months June, July, August, and September diarrhœa was very prevalent.

1899.	Dysentery Cases.	Diarrhœa Cases.
August 12th to 31st	—	3
September	1	11
October	3	3
November	2	6

Female Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	1	1
April	1	6
May	1	11
June	5	19
July	1	14
August	1	16
September	4	17
October	—	10
November	—	10

Ward D 1 accounted for 4 Dysentery Cases and 12 Diarrhœa Cases.
„ D 2 „ 3 „ „ 10 „
„ G 1 „ 1 „ „ 16 „
„ H 1 „ 1 „ „ 13 „

During the nine months 5 female attendants suffered with diarrhœa.

1899 (12th to 31st).	Dysentery Cases,	Diarrhœa Cases.
August	3	3
September	4	10
October	—	—
November	1	3

BANSTEAD ASYLUM.

Male Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	—	6
April	—	5
May	—	1
June	—	1
July	4	1
August	5	23
September	8	6
October	—	2
November	1	6

During the months of August and September a recrudescence of the disease occurred at Banstead. Ward K was particularly affected, no less than 8 out of the total 13 cases of dysentery coming from that ward; 4 cases of diarrhœa among the patients, and 4 among the attendants, were also reported for this ward within the same period. During the whole time, 9 cases of diarrhœa have been reported as occurring among the male staff.

Female Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	1	9
April	—	3
May	1	—
June	—	1
July	—	2
August	—	4
September	1	3
October	—	9
November	—	3

Only 3 cases of dysentery have been reported during the whole nine months, and the cases of diarrhœa have not been confined to any particular ward. No cases have been reported from the female staff.

COLNEY HATCH ASYLUM.

Male Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	1	1
Apriil	1	—
May	2	—
June	2	2
July	—	1
August	4	6
September	3	—
October	1	7
November	4	I

Ward C 5 contributes 5 Dysentery Cases and 4 Diarrhœa Cases.
„ 11 „ 6 „ „ — „

A male attendant from Ward 11 had an attack of diarrhœa.

The cases are not confined to any particular ward, but spread throughout the asylum.

1899.	Dysentery Cases.
August	9
September	—
October	—
November	—

Female Side.

1901.	Dysentery Cases.	Diarrhœa Cases.
March	3	2
April	4	—
May	—	2
June	7	3
July	6	5
August	5	7
September	10	7
October	6	20
November	3	17

For the nine months 19 cases of dysentery and 5 of diarrhœa have occurred in Ward 20. Except the case mentioned, the disease has not been confined to any particular ward, but spread throughout the wards.

Five cases of enteric fever have been reported.

During the nine months 5 cases of diarrhœa have occurred among the female staff.

1899.	Dysentery Cases.	
August 1st to 31st	11	No diarrhœa data.
September	7	
October	7	
November	1	

From the female side, during the period from March to November (inclusive), there have been reported 11 cases of enteric fever, which resulted in four deaths. Four of these cases came from Ward 23, 3 from Ward 17, the remaining 4 coming from Wards E2, 16, 26, 36 respectively.

On the three following pages will be found a Statistical Table, and two diagrams, illustrating the behaviour of Dysentery in Claybury Asylum from September 1st, 1898, to August 31st, 1901.

SHOWING RELATION OF DEATHS WITH DYSENTERIC LESIONS TO THE TOTAL DEATHS AT CLAYBURY ASYLUM.

—	September 1st, 1898, to August 31st, 1899.		September 1st, 1899, to August 31st, 1900.		September 1st, 1900, to August 31st, 1901.	
—	Male.	Female.	Male.	Female.	Male.	Female.
Total Deaths	118	144	116	136	79	100
Deaths with Dysenteric Lesions	27	50	14	32	7	15
	22.96 p. c.	34.7 p. c.	12 p. c.	23.5 p. c.	8.8 p. c.	15 p. c.
Total Deaths (Male and Female.)	262		252		179	
Deaths with Dysenteric Lesions (Male and Female.)	77		46		22	
	29.4 per cent.		18.2 per cent.		12.3 per cent.	

The above statistics probably represent the maximum, as the intestines were opened and carefully examined in every case. The dysenteric lesions refer not only to recent and active processes which in a large number of cases were the actual cause of death, but also to chronic, partially healed or entirely healed processes, affecting the large intestine. The post-mortem examinations for the last two years have been made by Dr. Bolton, and the comparative statistics of these two periods are therefore free from the fallacy of observations by different individuals.

CLAYBURY ASYLUM.

(SEPT. 1st, 1898—AUG. 31st, 1899.)

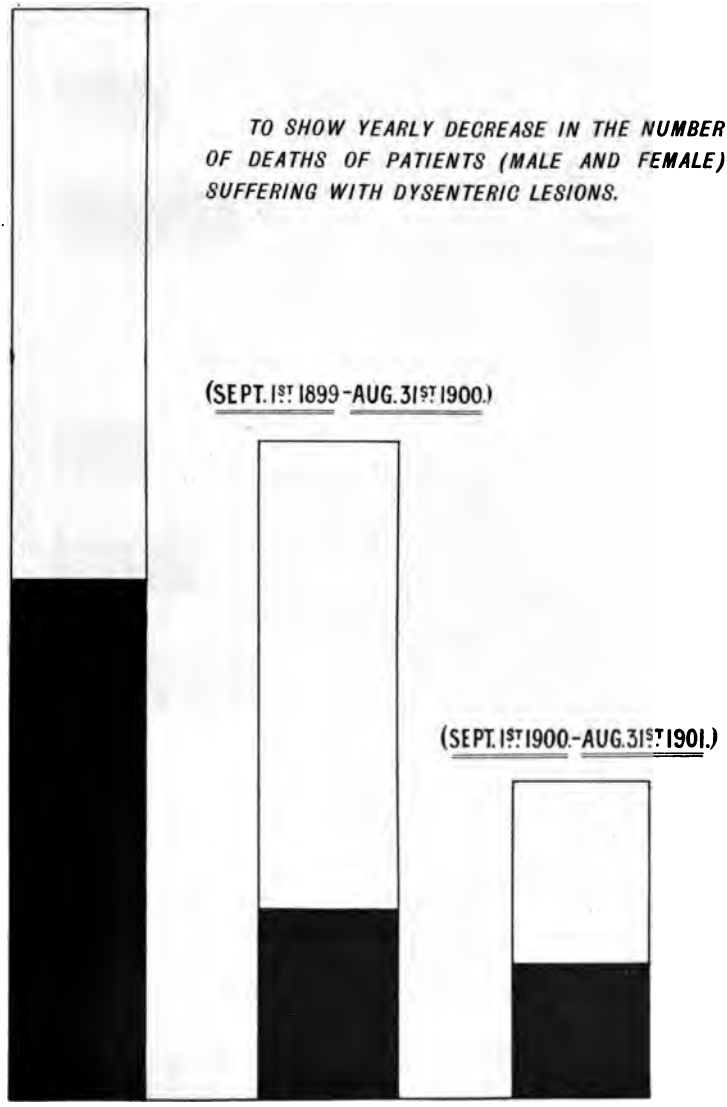
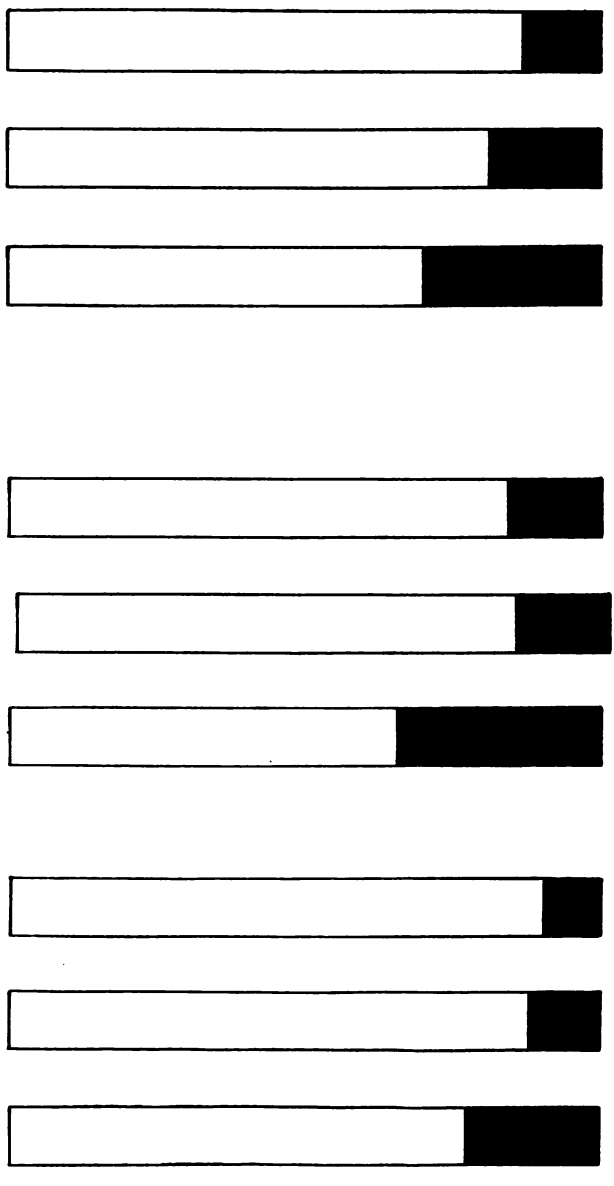


DIAGRAM INDICATES TOTAL NUMBER OF DEATHS.

Black Parts indicate death in recurrent cases.

Light Parts denote new cases.

SHOWING RELATIVE DECREASE OF PERCENTAGE OF DEATHS WITH DYSENTERY.



Discussion on Dr. Mott's Paper.

Dr. Claye Shaw: I have to thank the Epidemiological Society for giving me the opportunity of listening to Dr. Mott's Paper on Colitis. Why does Dr. Mott, I would ask, call his subject by a specific term, Asylum-Dysentery? There seems to be nothing peculiar in the disease he describes; nothing different from ordinary diarrhœa or dysentery, as seen in people living in large numbers under insanitary conditions. I can understand that what he describes is dysentery in asylums, but this does not imply quite the same thing as "Asylum-Dysentery." My Paper, read before the British Medical Association, was in answer to Dr. Mott's *Report*, and was intended to show that the Banstead Asylum (of which I was then superintendent) did not come under the conditions existing in some of the asylums described by him; and to point out that there was at least a possibility of another interpretation for what I take to be a condition inseparable from some states of chronic insanity, viz., a degeneration of the tissues of the intestinal coats (perhaps beginning in Meissner's plexus), leading to ulceration, and with diarrhœa or dysentery.

There is really very little between Dr. Mott and myself. I own that the condition he describes is "bacterial," and almost certainly infectious, and it is useless to deny that the conditions of sanitation at Claybury were insanitary; but my position is, that with the best conditions of nursing and sanitation in asylums there will still be ulcerations of the intestines, leading to diarrhœa, etc.; and that these lesions cannot be traced to anything but degeneration due to (most probably) nerve affections.

See how these conditions arise in asylums! They are scattered; they occur in broken-down and very degenerate cases; they do not occur (primarily) in the attendants; they come and go. The asylum may be free for weeks, and then, without apparent reason, one or more cases crop up. Of course, if the patients are not properly nursed the secondary effects will spread. The whole thing is something of a mystery. There are cases where are diarrhœa and dysentery, and yet no lesions are seen in the intestines after death; and there are cases where a large amount of ulceration is seen in various parts of the intestine, and yet there is absolutely neither diarrhœa nor dysentery. If we consider that many of these insane persons are degenerate from the most external layer of the epidermis to their

innermost drop of fluid, what is more likely than that the intestinal nervous and vascular systems should share in the degeneration?

Is not Dr. Mott's Paper premature in ascribing all conditions of diarrhœa and dysentery in asylums as due to inefficient nursing and sanitation? We require the experience of all asylums, public and private; and it would be advisable to know the conditions of the inmates of the workhouses.

At present the only thing established is, that under some circumstances an infective disease is set up, and that it can be brought within limits by nursing and by isolation. I beg to be allowed to recognise the great value of Dr. Mott's Paper, in calling attention to what is, without doubt, the true nature of much of the diarrhœa and dysentery that are found in asylums; but I think that he has only described the *preventible* form of it, and that there is still another cause that sets up the true "asylum dysentery," and that this cause is to be found in one of the factors of the insane state, viz., progressive intestinal degeneration.

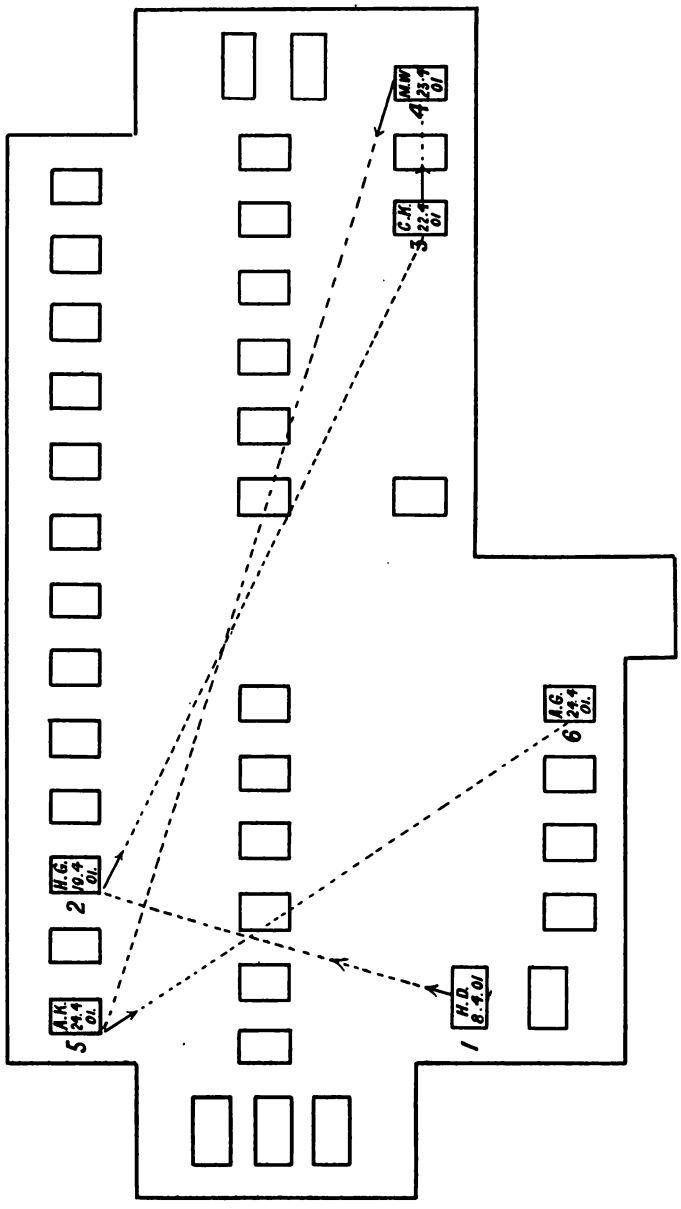
Dr. Percy Bailey, Hanwell Asylum, said: In the latter part of August, 1900, I became personally intimate with the symptoms of this condition. The onset was sudden, or at least was preceded by only a short period—a few hours—during which I felt "out of sorts." During this period there was nothing whatever to indicate the approach of any abdominal disturbance. My appetite had not been quite up to the mark during the day, and I experienced an uncomfortable degree of languor and a general desire to rest; but I had no headache or other pain. About eight o'clock in the evening I began to feel giddy, and on moving about was fearful lest I should fall. Shortly after this, my temperature in the mouth was 102 deg. I then went to bed, and slept until about 2 a.m. At this time violent diarrhœa set in, with headache and nausea, but no vomiting. There was now some abdominal pain and tenderness of a dull, gnawing, sickening, and depressing kind—not griping. When I again took my temperature I was rather astonished to find that the thermometer registered 104.8 deg. The diarrhœa continued to be most urgent throughout the following forty-eight hours, a motion occurring about every half-hour. At first the motions were free from blood or mucus, but both these became evident in the course of an hour or two. In my case, however, the blood was never very abundant,

although when the disease was fairly established (by the second day), the motions consisted only of mucus. After the onset of the diarrhœa the temperature rapidly declined, and did not rise above 99 deg. after the third day. At first there was no tenesmus, but this very soon became a most painful and troublesome symptom, and continued throughout the attack.

After five or six days I began to improve, and was able to leave my bed by the end of a week. My temperature was then normal, but convalescence was tedious.

My own case was typical of a class, but often the temperature does not rise to so high a point, and the symptoms may extend over a period of months. Some cases are heralded by frequent and very copious motions of the consistence of thin gruel, and containing so much blood as to be almost the colour of port wine. In these cases there is no doubt the small intestine is affected, as I have found this same material after death in that portion of the gut. I believe that this type of case is very fatal. Sometimes the abdominal tenderness is so great that the weight of the bedclothes is almost unbearable. Although in my own case there was no vomiting, this symptom is present in the vast majority, and may be most urgent and alarming. Sometimes, after a week or two, diarrhœa will cease, while the temperature is maintained at 100 deg. or higher. This, I believe, is a bad symptom, and indicates paralysis of the gut. In my experience it is followed by tympanites, perforation, and death.

Evidence of the infectious nature of the disease is accumulating, and a mild outbreak which occurred in Ward 15 (Female), in April, 1901, may be mentioned in this connection. It commenced with H. D., a very feeble old woman, who died April 18th, and had diarrhœa for ten days before her death. There was no post-mortem, and hers was not by any means a typical case of dysentery. On April 19th, H. G. (see plan of ward) was attacked. She occupied a bed some distance from that of H. D. On the 22nd, C. K., who was at quite the opposite extremity of the dormitory, was attacked: and on the 23rd M. W. Between the two latter patients only one bed intervened. On the 24th, A. K. and A. G. developed symptoms. In all six cases, out of a dormitory containing forty beds, five of these cases occurring within six days. With the exception of H. D., all these cases recovered. At the time of this outbreak there had been other cases scattered through the female wards, but they were of a sporadic kind. Fortunately, at this time on



the female side of the asylum we were enabled to carry out a fairly rigid system of isolation. The patients were

removed to the Isolation Hospital on and after April 15th. With the exception of C. K., M. W., A. K., and A. G. (all from Ward 15), no fresh case occurred in the female wards until April 30th. One case, however (L. M.), who developed diarrhœa on April 8th in Ward 8 (she occupied a single room), we could not remove, on account of her mental state (she was a very restless general paralytic); and on April 30th, as stated above, H. A. (Ward 9) developed the disease. She also, for similar reasons, could not be taken to the Isolation Hospital. After this the female wards were practically free from the disease (no fresh cases occurring) until July 6th.

COLONEL MACLEOD said that he had had a painful experience of the subject of Dr. Mott's paper while serving as Assistant Medical Officer in the Durham County Asylum, Sedgefield, during the years 1861-65, before he entered the Indian Medical Service. In the earlier years of his residence at Sedgefield there was no dysentery in the asylum; and when, in 1864, the disease broke out, it excited great interest and attention, and the cases were carefully investigated, clinically and pathologically. Curiously, in the same year there was a similar outbreak in the Cumberland and Westmoreland Counties Asylum, at Garlands, near Carlisle, the particulars of which had been very carefully recorded by the Medical Superintendent, Dr. T. S. Clouston, in a paper entitled "Sewage Exhalation the Cause of Dysentery," read before the Metropolitan Association of Medical Officers of Health on the 27th of May, 1865, and subsequently published in the *Medical Times and Gazette* (vol. i of 1865, pp. 567 and 597). This was an interesting and very able paper, which would well repay perusal. Briefly, the main points were, that the disease prevailed intermittently from February to August, 1864, and again in March, 1865; that in a population of 250, thirty persons were attacked, of whom twenty died; that two sane persons suffered; that the majority of cases (twenty-four out of thirty) occurred on the ground floor; that diarrhœa was unusually prevalent during the time among both patients and attendants; that several cases of typhoid fever occurred; that the cases appeared as a series of small groups; and that these outbreaks were associated with the irrigation of adjacent meadow land with putrid sewage. The clinical, pathological, and therapeutical aspects of the disease were fully detailed. The Sedgefield outbreak presented very similar features to the Garlands, and they were

also associated with sewage irrigation. On comparing the facts observed by himself and recorded by Clouston with those narrated by Drs. Gemmel and Mott, Colonel Macleod had no hesitation in concluding that the disease which occurred in these two asylums in 1864-65 was the same as that which still haunts English asylums. He had, in India and at Netley, had very large experience of tropical dysentery under a great variety of circumstances; and he held that, on clinical and pathological grounds, the so-called "ulcerative colitis" of English asylums was dysentery and nothing else. The bacteriological study of the disease was, he judged, not sufficiently advanced to furnish a certain instrument of comparison and differentiation between different forms of dysentery, and in different places and circumstances. He had, in India, found reason to associate the prevalence of dysentery with impure water and fœcal pollution, and he cited instances in which reforms in respect of purity of water and disposal of sewage had been followed by great reduction of prevalence and mitigation of type of dysentery, as well as of other bowel complaints. To the question, Why does dysentery, which has disappeared elsewhere, linger in English asylums? he would reply *more scotorum*, by asking why beri-beri broke out in certain asylums of England, America, France, and Germany, while it was unknown among the community at large. It seemed as if the infection in both cases found suitable victims in the debiliated denizens of mad-houses.

The evidence, as it stood at present, seemed to establish three conclusions, namely—

1. That the so-called "ulcerative colitis" of English asylums is dysentery.
2. That dysentery is caused by a fæcal noxa; and
3. That the prevention of dysentery depends, according to present knowledge, in taking all possible precautions to destroy this noxa, and to prevent its access to the healthy and, *á fortiori*, to the debilitated organism.

DR. E. W. GOODALL: The Society is greatly indebted to Dr. Mott for bringing before its notice a subject so deeply interesting to all who are engaged in the study of epidemiology. Dr. Mott is to be congratulated on the manner and thoroughness in which his work has been carried out, and the London County Council deserves our praise for having allowed and aided the investigation which has led to the results indicated in the paper we have met to discuss.

Since coming to this meeting, I have read Dr. Claye Shaw's observations upon Dr. Mott's paper; and from them I gather that he has practically abandoned the view he formerly held as to the nature of this disease. He admits it to be bacterial, and "almost certainly infectious." With respect, however, to the doctrine of its infectiousness, he is not yet apparently completely converted. To him the behaviour of the disease is still "something of a mystery." But as I read the sentences immediately preceding this confession, I am at once vividly reminded of another disease, concerning the infectious nature of which there can be no question—I mean diphtheria. Diphtheria has long been known to occur with some frequency amongst the patients convalescing from scarlet fever in the hospitals of the Metropolitan Asylums Board,* and also in other fever hospitals. With the exception, perhaps, of the sentence, "they do not occur (primarily) in the attendants," I might, with no essential alteration, apply Dr. Claye Shaw's remarks to diphtheria, as it has manifested itself in the institutions to which I refer. "See how this condition arises in fever hospitals! It is scattered; it occurs in those whose constitutions are weakened by an attack of another infectious disease (scarlet fever); it comes and goes. The hospital may be free for weeks, and then, without apparent reason, one or more cases crop up. Of course, if the patients are not properly nursed, the secondary effects will spread. The whole thing is something of a mystery." These words, I say, might with truth be applied to post-scarlatinal diphtheria in the fever hospitals. Yet no one denies that diphtheria is an infectious disorder. That this form of intestinal disease, so common in certain asylums, is infectious, has, in my opinion, been proved up to the hilt by Dr. Mott's researches. But between this disease and the secondary diphtheria to which I have alluded, there is one marked difference, concerning which I have not gathered any clear information from Dr. Mott's paper. Granting that both diseases are infectious, there yet remains the fact that, while diphtheria is of frequent occurrence in the community outside the fever hospitals, and may thus be introduced to them from without, this dysentery, on the other hand, is almost entirely confined to the asylums, and is very rarely met with outside. I would, therefore, ask Dr. Mott: How has the disease got into the asylums in the past, and how does it still continue to get in? With respect to the identity of this asylum disease with

* See *Transactions* of Epidemiological Society, New Series, vol. xv.

tropical dysentery, I can offer no opinion from personal experience. But I have occasionally seen isolated cases in hospital practice. One case of considerable interest I brought before the Hunterian Society, in March, 1899. A girl, sixteen years of age, was admitted to the Eastern Fever Hospital, suffering from enteric fever of a week's duration; there were rose spots, pyrexia, and moderate diarrhœa. The serum reaction (one in twenty) was positive. The patient complained of slight, but almost constant, abdominal pain, and there was occasional vomiting. These last-mentioned symptoms continued, and the patient emaciated. About five weeks after admission, the vomiting and pain became aggravated, and, in addition, frequent diarrhœa set in. Three weeks or so later, the patient died of peritonitis. At the *post-mortem* examination there was extensive ulceration of the large, and slightly of the small, intestine. This ulceration was not like what is seen in enteric fever, but was similar to that described by Dr. Mott in his severe cases. Perforation of the colon had occurred. The case appeared to be an exceptional one of mixed enteric fever and dysentery, such as those to which allusion is made by Dr. Mott and Dr. Durham in their report to the London County Council.

Dr. Mott has dealt with the disease as he found it in the London County Asylums. But there can be no doubt that it exists in other lunatic asylums, and possibly in other institutions. I have looked through the Annual Reports of the Statistical Committee of the Metropolitan Asylums Board for some years past, for the purpose of ascertaining whether there was any record of the disease occurring in their asylums. A tabulated list of the causes of death of the inmates is given; but, with the exception of one year at one institution, diarrhœal disease, apart from enteric fever, as a cause of death, is conspicuous by its absence. Dr. Mott believes that the disease is conveyed from one asylum to another through the transference of patients. If this view be correct, one would expect to find dysentery in the asylums of the Asylum Board, for both the Leavesden and the Caterham Asylums receive no small number of patients from the County Council asylums. The exception to which I have alluded is the Leavesden Asylum. In the summer of 1899, I was concerned, together with other investigators, in inquiring into the nature of an outbreak of fever in that institution. The cases divided themselves into four groups: (i) cases of enteric fever; (ii) cases of diarrhœa with pyrexia, neither clinically nor bacterio-

logically enteric fever; (iii) similar cases without diarrhœa; and (iv) cases of pneumonia, some of which had diarrhœa. A considerable number of fatal cases occurred, but in no single instance was there found a case that presented anything like severe ulceration of the large intestine. I suppose, therefore, that the diarrhœal cases were not of the nature of asylum dysentery. For it is difficult to believe that amongst so many cases (one hundred and forty to one hundred and fifty altogether for all the groups), not one should have presented the ulceration of the colon so characteristic of dysentery. The clinical symptoms, moreover, were not quite the same as those described by Dr. Mott; and, lastly, I may add that, contrary to the experience of the London County institutions, Dr. Arthur Shadwell, who had been entrusted with inquiring into the cause of the outbreak, definitely traced it to contamination of the water supply by sewage.*

I have stated previously that, with the exception of the year 1899, diarrhœa, dysentery, colitis, etc., as causes of death, were rarely to be found in the lists of deaths in the asylums of the Asylums Board. Yet, when we began our inquiry into the 1899 outbreak, we were told that outbreaks of diarrhœa had been common, and mostly occurred in the spring and autumn; and that the disease was even fatal. Apparently, as long as the affection was considered to be only diarrhœa, even though it was fatal, it did not matter very much; but directly the word "typhoid" was breathed there was alarm and inquiry.

It is to be hoped that Dr. Mott's paper will engage the attention of public bodies throughout the kingdom: that it may be ascertained definitely whether the prevalence of this disease is wide, and whether it occurs in institutions for the sick and infirm other than asylums.

South African Dysentery, and its relation to Asylum Dysentery. By Dr. Owen Richards and Dr. Washbourn, C.M.G.

DR. OWEN RICHARDS and DR. WASHBOURN stated in their joint communication that the dysentery met with in South Africa closely resembles, in its clinical aspect and morbid anatomy, that of Asylums.

Clinical Course.—The principal feature of the disease is the absence of fæcal matter from the stools, and its replacement by blood and slime. This is accompanied by tenesmus, frequent calls to stool, and abdominal pain.

On examining the evacuations under the microscope,

* See Report of Statistical Committee of the Metropolitan Asylums Board for 1899, vol. ii, pp. 115, 181, and 186.

they are found to consist of red blood corpuscles, intestinal epithelium, and many polynuclear leucocytes.

In one type of the disease there is little or no blood and slime, the bowels being either constipated or loose, and the abdominal symptoms not constituting a prominent feature of the disease.

Types of Disease.—The disease may be divided into three main types: although these types are connected together by a large number of intermediate types.

Type I.—This is the usual form taken by the disease.

It is characterised by a febrile stage, lasting about a week. The pyrexia is of an irregular character, varying from 101 deg. to 104 deg., with morning remissions and evening exacerbations. In addition to the abdominal symptoms and the usual symptoms of fever, a special symptom is the coating of the tongue with a thick, white, moist fur, looking like a layer of paint, on the surface. The attack may end in recovery or in death.

(*a*) The usual termination is that the temperature goes down in about a week, and at the same time the blood and slime disappear from the motions, and fæcal matter reappears. In a few days the patient is convalescent.

(*b*) Some cases become chronic. After the temperature has gone down, diarrhœa, with or without blood and slime, persists for weeks. The diarrhœa may alternate with constipation. The patient becomes very emaciated, the abdomen retracted, and the tongue red and glazed. He may ultimately recover, or die from exhaustion. Not infrequently, during the course of a chronic case, the patient suffers from distinct relapse, accompanied by the pyrexia and other symptoms of the primary attack.

(*c*) Death may occur within a week during the febrile stage.

(*d*) Death may occur some days after the pyrexia has subsided, the other symptoms continuing.

Not infrequently the actual cause of death is perforation of the intestine and consequent peritonitis. This occurred in three out of nine deaths at Deelfontein.

Type II.—These are mild cases without a febrile stage, which rapidly recover.

Type III.—In this type the principal feature is fever, of a more or less continuous character, lasting for some three weeks.

The symptoms closely resemble those of enteric fever,

and in one case seen by the writers of this paper, the diagnosis of enteric fever was made during life.

In some of these cases the attack begins with the passage of blood and slime; but in other cases blood and slime are absent during the whole course of the attack, the bowels being either loose or constipated.

These cases resemble the typhoid type of scarlet fever, and are probably due to the same cause, namely, a secondary septic invasion.

Mortality.—The mortality of South-African dysentery is low. For instance: of 379 cases at the I. Y. Hospital in Pretoria, there were seven deaths; a mortality of 1.86 per cent.

Complications.—Liver abscess is decidedly rare. In one case which was carefully examined, the pus was found full of streptococci and bacilli, thus differing from the usual solitary abscess after tropical dysentery.

Morbid Anatomy and Pathology.—The large intestine is the part affected, although in some cases the lower part of the ileum is also involved. The cœcum and rectum are the parts most affected, and in some cases the intermediate portions are free. The chief lesion is a great thickening of the walls of the intestine, accompanied by hæmorrhages. In all the cases we have seen there has also been ulceration, which has varied from a few small rounded ulcers to extensive honeycombing of the bowel.

Perforation of the bowel occurs not infrequently.

We have no knowledge of the pathology of the disease. We know, however, that amoebæ are absent, both from the intestinal contents and from the pus in cases of liver abscess. In many cases we have made most careful microscopal examinations of the intestinal contents.

Treatment.— We are satisfied that treatment with Ipecacuanha, or with magnesium sulphate, when *properly carried out,* is of great value.

Infectivity.—It is difficult in a military hospital, where the patients are frequently moved from one tent to another, to trace infection. Without having definite proof, we are nevertheless of the opinion that the disease is infective in the same sense as is enteric fever.

Relation to Asylum Dysentery.—From the above account it will be seen that South-African Dysentery closely resembles Asylum Dysentery. It principally differs in being

less severe. This can be accounted for by the difference in the vitality of the patients attacked; and in this connection we have noticed that patients convalescing from enteric fever, when attacked with dysentery, suffer much more severely than previously healthy individuals.

We have not seen cases dying before the stage of ulceration, which can be accounted for in the same way.

DR. ROLLESTON stated that his experience of asylum dysentery was limited to those sporadic cases admitted into general hospital, which were usually chronic and very rarely acute. He had seen South-African Dysentery at the Imperial Yeomanry Hospital, Pretoria, where he had succeeded Dr. Washbourn. The two diseases had a general resemblance clinically, and neither of them was amœbic in origin. When hepatic suppuration followed South African-Dysentery there were multiple abscesses on the liver like those seen on pylephlebitis, but there was no suppurative thrombosæ of the portal vein. Single hepatic abscess was not a sequel of South-African Dysentery, and the multiple liver abscesses were uncommon. The question as to the identity or independence of Asylum and South-African Dysentery could only be decided bacteriologically; until this was achieved surmises only could be made. Sections of the colon from two acute cases of South-African Dysentery seemed to show that the inflammation began in the mucous membrane and involved the submucosa secondarily. It was undesirable to generalise from such small data; but if it turned out that this was the rule in South African Dysentery, it would constitute a difference between African and Asylum Dysentery. For Dr. Mott pointed out that in the latter the submucosa was primarily affected. While bacteriological evidence was wanting, it was well to consider whether the features common to the two diseases were more striking than would result from the same organ of the body being attacked by acute inflammations due to different causes. In other parts of the body the results of acute infections, which could now be differentiated, had many points of resemblance: for example, in broncho-pneumonia the signs and symptoms were fairly constant, though the condition might be due to different infections.

Dysentery at Aldershot.

DR. T. DUNLOP: It will doubtless be of interest to hear of an unusual epidemic of dysentery, which made its appearance in Aldershot during August and September of last year (1901).

My attention was first drawn to its appearance by the weekly return of the Registrar of Deaths, showing that three deaths had occurred in one house on July 27th. Another occurred on August 2nd. There was then a lull till August 17th, but from that date till September 9th, when the epidemic was at its worst, there was a succession of fatal cases.

The disease in most instances started with ordinary diarrhœa, followed by a discharge of blood and mucus (described by some as consisting of flesh and blood). There was not much abdominal pain, but extreme tenesmus. In some cases, a condition of semi-coma was present. None of the medical men attending appear to have noticed much rise of temperature. Medical practitioners who had resided here for many years had never previously seen an outbreak of this kind in Aldershot. Several medical men familiar with the tropical form of the disease, Dr. Washbourn and Lieut.-Col. Franklin, R.A.M.C., who had experience in South Africa, and Dr. Donald, in Uganda, when in medical charge of the Indian troops serving there, confirmed the diagnosis of dysentery.

The results of inquiries as to the probable cause of the disease may be epitomised as follows:—

Fruit.—There was little evidence that this was a cause. Many patients deny eating fruit at all.

Milk. — The infected houses were supplied from a variety of sources, and many families only used condensed milk.

Water.—No cases occurred in the camp, which is supplied from the same source. A bacteriological examination of the water showed nothing abnormal.

Insanitary Surroundings.—Although in some houses insanitary conditions were found, yet in the majority of houses there was no reason to suspect the drainage. Also, certain portions of the town which have not got a reputation for cleanliness, escaped entirely.

Overcrowding.—In only one case was I able to obtain evidence of overcrowding.

How the disease was first introduced is a mystery. Being unusual, I naturally thought of the possibility of its being introduced by invalids who had returned from

South Africa. Although this is most probable, yet it has been impossible to directly trace it to this source. From inquiries made from the Medical Officers of Health of about twenty-four different districts where troops are stationed, I found that in none of them had there been any unusual prevalence of this disease. Only one case of dysentery occurred in the Camp, and this did not prove fatal.

However the disease was first introduced, there is not the slightest doubt it spread by contagion. Numerous instances occurred of parents being attacked after nursing their children, and relatives of patients living in different parts of the town attacked after visiting. In one house two children died from disease, and their parents were attacked; a third child was at once removed. She remained quite well for ten days, and as her parents were convalescent she came to the house with a paper. Whilst there she played with some water, said to be the fourth washing of infected clothes; within forty-eight hours that child developed dysentery. In some instances no evidence of direct contagion was obtainable, in these cases I believe the disease was spread by flies, which were very numerous this summer.

From inquiries made of the various medical practitioners, I estimate that there were between 170 and 180 cases during the outbreak. Of these 38 proved fatal. This gives a mortality of about 27 per cent.

The sex and age at death are as follows.—

	Under 1 yr.	1—5.	5—15.	15—25.	25—65.	Over 65.	
Males ...	2	12	2	1	0	2	
Females ...	6	8	1	2	1	1	
	8	20	3	3	1	3	= 38.

During the same period sixteen deaths from ordinary summer diarrhœa occurred.

During the whole of the year 1900 there were twenty-nine deaths registered from diarrhœa.

Aldershot Population Census, 1901.

Civil	16,726
Military	14,248
	30,974

Clinical Notes upon Dysentery at Aldershot

DR. E. J. F. HARDENBERG: While doing a *locum tenens* at Aldershot this summer I had nineteen cases of dysentery under my care, the details of which I am able to communicate to you, thanks to the courtesy of Drs. Stroyan and Gibson.

Of the nineteen patients, twelve were children, of whom three died, and seven were adults, the oldest of these being seventy-one, all of which recovered. In most of the cases more than one inmate of the house was attacked, the second one being taken ill two to nine days after. All my patients lived in one neighbourhood, but the condition of the houses varied greatly: some of the houses being in a filthy condition, with practically no means of nursing, while a few were scrupulously clean, and everything that could be done in a small private house in the way of preventing contagion was accomplished. For instance, the motions were passed on diapers, which were immediately burnt after being soiled, and all soiled clothes were allowed to soak in disinfectants—as Creolin, Sanitas, and Jeyes—and hands were washed after cleansing patient; but still in these houses another inmate got dysentery, although not so severely.

The onset of most of the cases was quite sudden, although in some cases the patients had had some diarrhœa for a few days previous; but this was not bad enough to confine them to bed.

All the patients passed at first very frequent stools, some passing twenty or more in the twenty-four hours. The stools during first days were of very small quantity, and consisted of blood, glairy mucus, and pus, and were of very foul odour, and no fœcal matter was passed. After about a week the motions diminished in number, the blood and pus became less, and the motions became more truly fœcal in character, but still containing quantities of mucus.

The attacks were accompanied with great tenesmus, both during the passage of the motions and independently. The tenesmus was likened by one adult to labour pains, and the cries of the children, in some cases, reminded one of the meningeal cry.

In nearly all of the patients some pyrexia occurred, the highest being in one child who had a temperature of 103 deg. The temperature mostly subsided after four or five days, and was then either normal or subnormal. The falling of the temperature mostly coincided with diminution of the symptoms, although a few patients, while still very ill, had subnormal temperatures.

The abdomen in the severer cases was retracted, or normal in appearance, but in no case did I find any distension.

The patients complained of pain in the abdomen, which was either referred to the umbilicus or to the region of the transverse colon.

The tongue mostly was furred, but not dry.

The collapse of the patients who were severely attacked was marked, and in the cases of the three children who died they were semi-comatose for the last four days, except to utter cries when tenesmus occurred.

There was wasting in practically all of the patients, and in one case a strong, healthy labourer lost over two stone during the fourteen days he was ill. There was practically no vomiting, although there was some nausea.

I noticed no complications in any of the patients. In the three children who died, their deaths appeared to be due in all three cases to collapse; one of the little boys had quite stopped passing blood, and was only passing very little mucus.

The duration was mostly fourteen days, but in one or two of the milder cases there were no symptoms after a week. One child died within four days of my first seeing it, and the other two lived a week.

I am sorry to say that I was unable, in either of the three patients that died, to procure a post-mortem examination.

With regard to treatment, the patients were fed on milk, milk and barley water, milk and lime water, and some of the severer ones on albumen water alone. During the stage of collapse they were given small doses of brandy or whisky. In my earlier cases I gave the patients astringents: as tincture of catechu, dilute sulphuric acid, aromatic chalk, and small doses of tincture of opium.

Some of the patients were given powdered ipecacuanha, the children being given four or five grain doses three times a day, and the adults about six grains three times a day. I was afraid to give larger doses, as there was great difficulty in many cases of nursing and the people carrying out instructions; and, moreover, I could myself give them only very little attention, as the neighbourhood was over a mile away, and I was very busy at the time. All the patients who were given ipecacuanha stood it well, and had no sickness afterwards, although some had some nausea. One of the children who died the ipecacuanha

apparently stopped the dysentery, but the child died, as I have already mentioned, of collapse.

The latter cases I treated with magnesium sulphate and dilute sulphuric acid, the adults having ℥j of mag. sulph. and 10 min. of dilute sulphuric acid four-hourly, till the blood and mucus stopped, and then astringents were given.

I obtained by far the best results from the treatment by magnesium sulphate. The dysentery in the old man of seventy-one stopped in five days.

COLONEL DAVID BRUCE, R.A.M.C., had, he stated, had practical experience in a laboratory in South Africa for three months, and during that time out of 30 cases examined, amœbœ had been found in one case only; indeed, he had grave doubts as to the existence of amæbic dysentery. He had isolated many organisms of the "coli" group, but he had not discovered a bacillus which could be regarded as the cause of the disease.

THE PRESIDENT (Dr. Patrick Manson) pointed out that the evidence as to the prevalence of Dysentery in asylums was altogether overwhelming, and he would like to ask whether such prevalence of the disease had been recognised by His Majesty's Commissioners of Lunacy, and, if so, what step they had taken to limit its spread? The London County Council were, he thought, much to be congratulated upon their public-spirited policy in publishing Drs. Mott and Durham's *Report*, and he trusted that the same spirit would obtain with regard to carrying out their recommendations. He ventured to question the desirability of creating vast asylums where the sick were aggregated together, and he thought it very difficult for one man to superintend such gigantic institutions, and at the same time to pay sufficient attention to the medical aspects of the situation. The strict discipline of attendants was most essential to proper administration, and the notification of Dysentery should be rigidly insisted upon. He thought it rather a strange policy to instruct the attendants in psychology and to disregard the elements of sanitation.

THE SUPPOSED INFECTIVITY OF DESQUAMATION IN SCARLET FEVER.

By C. KILLICK MILLARD, M.D., D.Sc.;
Medical Officer of Health and Medical Superintendent of the Borough Isolation Hospital, Leicester; late Medical Superintendent, City Fever Hospital, Birmingham.

(*Read: February 21st*, 1902.)

One of the many points in connection with the natural history of scarlet fever calling for further investigation, is the supposed infectivity of the desquamation which is so marked a feature of the disease.

From the point of view of fever hospital administration, the question is one of the first importance, for the prolonged detention of scarlet fever patients, which is still enforced in so many hospitals, is largely out of deference to the prevailing belief that the presence of any trace of desquamation is evidence of continued infectivity.

The view that desquamation is infectious was authoritatively endorsed by the committee appointed by the Clinical Society of London, "to investigate the periods of incubation and contagiousness of certain infectious diseases." Their report was published in 1892, and in their conclusions as to scarlet fever they state: "(2) . . . Infection certainly persists so long as there is any desquamation, and may still be active at the end of eight weeks."

The evidence, however, upon which this emphatic and unqualified statement was presumably based was of the scantiest and most inconclusive description. Out of 209 cases received in evidence in connection with scarlet fever, *only* 3 had the slightest bearing upon the question of the infectivity of desquamation. Indeed, I think I am right in saying that only in these 3 cases was the question of desquamation even referred to.

These cases were as follows:—

"Case XXIV (communicated by Dr. Murchison in 1878).

"On April 1st, 1878, Master P. left school at Wimbledon, on the fortieth day of an attack of scarlatina, for his home at Brighton.

Before leaving, desquamation had to all appearances quite terminated, the feet having desquamated twice. Also, he had had repeated carbolic acid baths, and he had left all his infected clothes behind. After reaching Brighton, his face and feet desquamated again, and four days after his arrival his mother fell ill with scarlet fever."

"CASE 58 (communicated by Dr. Whitelegge).

"B., a girl aged 19, slept one night with a cousin who was undergoing a second desquamation on the feet eight weeks after the original attack. B. had scarlet-fever rash four days subsequently."

"CASE 64.

"A girl desquamating after scarlet fever went to live in a household, and a boy aged 5 years had scarlet-fever rash five days later."

The details given in the above cases are so meagre that it is quite possible that there may have been a simple explanation of the conveyance of infection quite independent of the desquamation. It is unnecessary to assume this, however, because it is well known that infection is often carried after prolonged periods of isolation, when there is no discoverable lesion whatever. The committee themselves admitted this, for they stated: "It is, however, well shown . . . . that a patient who is completely convalescent—so far, at any rate, as would appear on a cursory examination—may yet be, in some way, capable of infecting others."

Whilst fully recognising the very valuable and scientific nature of their report as a whole, it is difficult to avoid thinking that the committee's "conclusion" was, in this case, rather the expression of a preconceived opinion than a genuine conclusion arrived at from the evidence examined.

Whether or not, however, the committee were justified in coming to the conclusion they did, there can be no doubt that they were supported in the view they took by the vast majority of the medical profession.

The Hospital (Nov. 7th, 1896), in an editorial said;—"In the good old times, the good old simple plan was to go by the "peeling," and maintain that so long as there was desquamation there was danger; and that if a patient who had finished peeling still appeared to carry infection, there must have been a mistake somewhere. Probably this is the opinion of nine out of ten doctors even now."

Although this was written over five years ago, I believe it still holds good to-day—so far at least as general practitioners are concerned.

So firm is the belief that a desquamating patient is

necessarily still infectious, that actions at law have repeatedly been based upon it as if it were a settled fact. Parents have frequently been prosecuted by corporations and convicted; and, on the other hand, corporations have been sued by parents and mulcted in heavy damages. A good example of the latter was the case of *Keegan* v. *the Corporation of Birmingham*, which occurred in 1896. A patient was sent home from the Birmingham City Hospital at the end of six weeks, still desquamating—a written note of this fact being made at the time. Scarlet fever again broke out in the family; several members contracted it, and one died. The jury gave a verdict for the plaintiff, awarding £50 damages, the full amount claimed; whilst adding a rider, however, exculpating the medical officer who discharged the patient. The most glaring example of vexatious litigation in connection with this subject is probably that of *Jameson* v. *the Metropolitan Asylums Board*, which also occurred in 1896. In this case, according to the report which appeared in the press, a claim was made against the Metropolitan Asylums Board by the secretary of a training school for girls for £13 for expenses in consequence of one of the girls being sent back from hospital still desquamating. The expenses were incurred owing to some of the boarders having to be sent home, and the whole house having to be disinfected over again. No one else contracted the disease, but a general practitioner had given it as his opinion that the girl was still infectious. The facts proved in evidence were that the patient was discharged from Winchmore Hill after being in hospital sixteen weeks! Seventeen days later, she was found to be desquamating on the fingers and toes, and was returned to hospital the following day. She was then isolated for a further period of six weeks.

The judge said that it was impossible to say that there was carelessness on the part of the hospital doctors, and so gave judgment for the defendants. At the same time he refused costs, on the ground that it was *a proper case for inquiry*.

In the face of such cases as these, medical superintendents of hospitals, whatever may be their own convictions, find little encouragement to discharge patients from hospital until all trace of peeling has disappeared. And so the old belief is fostered, and our search after scientific truth is checked.

Believing very strongly myself that the old view was not supported by experience, and that it required modify-

ing, if not reversing, I decided to ascertain the state of expert (as opposed to popular) opinion on the subject.

I accordingly drew up a letter, a copy of which I sent to the medical superintendents of the principal fever hospitals in the country, and to a few others whose opinion I believed would command respect—twenty-five in all.

The letter was worded as follows:—

TOWN HALL,
LEICESTER,
November, 1901.

DEAR SIR,

The Infectivity of Desquamation in Scarlet Fever.

Considerable uncertainty and difference of opinion exists at the present time as to the importance which should be attached to the process of desquamation after scarlet fever as a source of infection.

On the one hand, there is the old and still very generally accepted view that the particles of desquamating epithelium are, *per se*, highly infectious, and that no patient after suffering from scarlet fever can be regarded as free from infection so long as any trace of this process on any part of the body persists.

On the other hand, there is the more modern view, which holds that there is no evidence to show that the particles of desquamating epithelium are infectious, *per se*,* and that there is good reason to believe that the process of desquamation may, and frequently does, continue for a considerable time after a patient has ceased to be infectious.

The issue is one of great practical importance in the administration of fever hospitals; for, if the latter view be correct, it follows that it is not necessary to continue isolation long after the usual period, merely on account of protracted desquamation.

For example, it not infrequently happens in the case of adults that the process of desquamation, even after a slight attack, may not be completed—more especially on the soles of the feet—at the end of seven or eight weeks, although the patient may appear, in all other respects, to have quite recovered at the end of five or six weeks. The additional two or three weeks' detention in hospital—required if the old view be correct—not only increases the already heavy cost of hospital isolation, but is often a great inconvenience both to the hospital authorities, whose accommodation may be limited, and to the patient himself.

It has occurred to me that it might tend to elucidate the question at issue if the views of those who, from special experience, should be best able to form an opinion, were collected with the object of publication.

Moreover, if it should appear that there is a considerable body of expert opinion inclined to the view that prolonged desquamation after scarlet fever is not necessarily the indication of danger which

* Of course, whilst a patient is still infectious the desquamating cuticle may act as *fomites* and carry infection, just as the clothing worn by such a patient may. This is quite a different thing, however, from the cuticle being itself the source of infection, as in the former case its mere presence would not be evidence that the patient was still infectious.

tradition has taught the public to believe, it will facilitate the work of those engaged in the administration of fever hospitals who may find themselves hampered by such tradition.

I shall be much obliged, therefore, if you will kindly do me the favour of replying to such of the enclosed questions as you feel able to answer.

I am, dear Sir,
Yours very faithfully,
C. KILLICK MILLARD.

Twenty-one of these gentlemen have very kindly favoured me with replies to my questions, and these will be found tabulated in Appendix I. I have divided them into two groups :—

I.—Those taking what I will call the "new" view (though I recognise, of course, that it has no real title to the designation),

II.—Those adhering to the old view, or occupying an intermediate position.

The uniformity in the answers in group I, shows, I think, that the points at issue, as indicated by my letter, were well appreciated. I must confess I was surprised to find so much uniformity.

Practically, all the gentlemen in group I, sixteen in number, state that :—

1. They can adduce no evidence that desquamating epithelium is, *per se*, a source of infection.

2. They consider that too much importance has in the past been attached to desquamation as a source of infection.

3. Their experience does *not* support the popular view that desquamation after scarlet fever is necessarily an indication that a patient is still infectious.

4. They believe that a patient may continue to desquamate for some time after he has ceased to be infectious.

5. They do not believe that it is necessary, in order to prevent the spread of infection, that patients, who otherwise are quite ready to leave hospital, should be detained until every visible trace of desquamating epithelium has disappeared.

There is much less uniformity in the answers in group II. They are only five in number, and one or two may almost, I think, be said to favour the new view.

Judging from the answers I have received, therefore, it is clear that a great preponderance of expert opinion is almost directly contrary to the old and popular view. If expert opinion be right, it follows that the existing

practice of detaining all patients till the last trace of desquamation has disappeared, which is based on the old view, could be safely modified in the direction of considerably shortening the period of isolation, to the great advantage of everyone.

It will be well, therefore, to consider what are the arguments in favour of and against the old view.

I.—In favour of the old view :—

(*a*) *Tradition.*—It is certainly an old-established and widely-accepted tradition that the desquamation of scarlet fever is highly infectious. It is easy, however, to understand why this should be so. Scarlet fever is a disease in which one of the most obvious and striking characteristics is the desquamation of the cuticle : a process prolonged in many cases long after convalescence is established and all other evidence of the disease has disappeared. Scarlet fever happens to be a disease in which the duration of infectiousness is also often prolonged long after convalescence is established. What could be more natural than to assume that these two characteristics stood in the relationship of cause and effect!

(*b*) *Apparent Confirmation of Tradition by Experience.*—It has frequently happened that patients still desquamating have carried the infection of scarlet fever to others, as in the three cases reported by the committee of the Clinical Society. Probably almost every practitioner has met with similar cases. In the absence of proof to the contrary, and with the importance of such conditions as an inflammatory condition of the nasal mucous membrane not then recognised, it is scarcely surprising that *post hoc* should have been assumed to be *propter hoc*, and that such cases should have been accepted as evidence of the correctness of the old tradition. It must, I think, be admitted by all scientists that evidence of this description can only be regarded as reliable if supported by carefully-compiled statistics, which at present is not the case.

(*c*) *The Belief that Scarlet Fever is more Infectious during Convalescence than during the Acute Stage.*—If this were really the case, it would seem to give some support to the old view. It is probable, however, that the belief is erroneous.

It occasionally happens, it is true, that in a household where one child has been attacked by scarlet fever, the other children may escape during the first few weeks and then contract it. It is such instances which have almost

certainly given rise to the above-named belief. These cases can easily be explained, however. The period of infection in scarlet fever is undoubtedly a long one, and continues after the patient becomes convalescent and is able to get up. During the acute stage, while the patient is in bed, isolation from the rest of the family is comparatively easy; subsequently, however, it is much more difficult and is apt to break down,

Moreover, it is known that infection which is resisted at first may be contracted after prolonged exposure. This is repeatedly observed in fever hospitals, where new members of the staff, or cases admitted through erroneous diagnosis, may not contract the disease until after an exposure of several weeks. The fact that the duration of infection is prolonged in scarlet fever is clearly quite independent of desquamation, for it certainly begins prior to the process of desquamation and frequently continues long after all traces of desquamation have disappeared. It is important to bear this in mind.

(*d*) *Apparent Analogy to Small-pox.* — The scabs in small-pox are known to contain the infecting elements of that disease; and at first sight the desquamation of scarlet fever would appear to be somewhat analogous to the scabbing of small-pox. The analogy, however, is only apparent, and if examined closely will be found to go little further than the fact that the two diseases are both zymotics. A comparison with small-pox, indeed, is really against the supposition that the process of desquamation is infectious, and it will be referred to again.

The above are all the arguments in favour of the old view which I have been able to discover. Of direct evidence, either statistical, clinical, or pathological, there appears to be none.

II.—*Against the Old View that the Desquamating Epithelium is Infectious.*—To prevent any misconception, I wish it to be clearly understood that, in suggesting that the desquamation of scarlet fever is not infectious, I simply mean that it is not infectious *per se*. The flakes of epithelium given off by a patient still in the infectious stage may naturally be expected to carry the infection as *fomites*, just as would the clothing worn by such a patient, or other articles in contact with him. On this hypothesis, a piece of desquamated cuticle from a scarlet-fever patient, if sent in a letter through the post might readily convey the disease, if the patient from whom it came was still in an

infectious state; but the mere presence of desquamation ceases to be any evidence that a patient *is* still in an infectious state. The real question at issue is—Does the desquamating epithelium contain in itself the infecting elements, *i.e.*, the supposed germs of scarlet fever, in the same way that the scabs of small-pox are believed to do?

The arguments, then, are:—

(*a*) *The Comparative Absence of Positive Evidence Supporting the Old View.*—This, I hold, is a strong argument against the old view. It is difficult to believe but that, if desquamation were really infectious, strong and direct evidence of the fact would ere this have been forthcoming.

On the other hand, there is some direct evidence against the old view.

(*b*) *Failure to find in the Desquamating Cuticle the Streptococcus Conglomeratus.* — The *streptococcus conglomeratus* of Klein has probably the most claim at the present time to be regarded as the specific organism of scarlet fever. Should this claim be finally substantiated, the fact that it is not present in the cuticle would be highly significant.

(*c*) The next consideration is one to which I attach considerable importance. The "striking distance" of scarlet fever is a very short one, as is proved by the well-established fact that a properly-administered fever hospital may be placed in the midst of a crowded locality without risk of spreading the disease. But if the desquamating cuticle contained the germs of the disease, it is almost inconceivable that such could be the case, for the physical character of the flakes of epithelium, whilst protecting the germs from the disinfecting and attenuating action of the atmosphere, admirably adapts them for being carried long distances by currents of air.

Through the open windows of convalescent fever wards, innumerable particles of desquamating cuticle must be carried away by every breeze, to be scattered broadcast amongst the surrounding population. If the desquamating cuticle is so highly infectious, how is that no harm results from this?

(*d*) *The Comparison between Scarlet Fever and Small-pox.* —This, as I have already stated, although at first sight it might seem by analogy to support the view that desquamation is infectious, in reality tells against it.

We know that the scabs of smallpox are highly infectious. But then the eruption of small-pox is pustular, and pustulation is a process essentially characteristic of microbic action. The contents of the pustules undoubtedly contain the specific microbes of small-pox, and it is only to be expected, therefore, that the scabs, the remains of the pustules, should also contain them. This provides a simple explanation of the fact that the "striking distance" of small-pox is such a long one, and that its infection is so readily air-borne.

In scarlet fever, however, the eruption is an erythema—a process characteristic of chemical poisoning, or of poisoning by the chemical products of microbic action, rather than of the direct action of microbes themselves. Thus we have erythematous rashes, closely resembling that of scarlet fever, after the use of certain drugs, and in cases of septic absorption; and these erythemata are sometimes followed by desquamation. Desquamation, much more profuse even than that seen in scarlet fever, follows the eruption of *Erythema scarlatiniforme desquamativum;* yet there is no reason to think that the process in any of these cases is a microbic one. I hold, therefore, that the desquamation of scarlet fever should be regarded as also a non-microbic one.

(*e*) In the absence of bacteriological evidence, it appears to the writer that the only way in which this question can be settled is by specially-collected statistics.

A number of cases must be sent out from hospitals still desquamating, but otherwise apparently quite free from infection, and the result compared with that of similar cases in which desquamation has been completed. At present, those responsible for the discharge of patients from fever hospitals are greatly hampered by the existing tradition as to the infectiousness of desquamation. It will be necessary, therefore, in order to get a sufficient number of cases, first to modify public opinion—which is one object of this paper—and then for some hospital or hospitals to take up the matter openly and avowedly as an experiment. In a small way, I have myself attempted something of the kind, both in Birmingham and in Leicester. My object was to find out whether patients sent out desquamating were more frequently followed by "return cases." My records in Birmingham covered some 4,810 cases, during the two-and-a-half years, 1894-96. 8.0 per cent.* of these

* This percentage was estimated from the records of only 2,500 of the cases discharged, but the same system of discharging was followed throughout.

were noted as showing some trace of desquamation (principally late desquamation on the feet), at the time they were discharged. But of the cases which carried out infection (by giving rise to "return cases"), only 6.5 per cent. had been noted as showing traces of desquamation.

These figures prove, I consider, so far as they go, that patients sent out desquamating do *not* give rise to any increased proportion of "return cases." Unfortunately, the collection of statistics of this nature involves—as I know from experience—considerable time and trouble; but it is very desirable that they should be collected on a much larger scale than I was able to do. I am in hopes that the Metropolitan Asylums Board, with their vast machinery, may one day have some such statistics available.

In Leicester, during the three months, September 14th to December 14th, 1901, I have been trying the experiment of reducing the minimum stay in hospital to four weeks. The average stay in hospital, including time ill at home, during these three months, has been 39.1 days, or, counting only time in hospital, 35.3 days (excluding* cases which were sent home on account of erroneous diagnosis).

In order to obtain this result, it was necessary in many cases to "pass" patients prior to completion of all desquamation. The total number of cases sent out was 190, and the number of "return cases" was five. The particulars of these "return cases" are given in Appendix II. In only one case was there any desquamation present in the "original" or "infecting" case: this was on the feet, and it was also noted that the patient was suffering from a sore nose, which latter fact alone is, in my experience, amply sufficient to account for the "return case."

An attempt to send patients out of hospital at the end of a month is not, in the present state of public opinion, free from difficulties. There is always, of course, the possibility that "return cases" may occur with patients sent out desquamating; and, should this happen, censure is likely to follow. A medical man is called in, and if he finds the least trace of peeling he frequently expresses the opinion that the patient was sent out from hospital too soon. In the great majority of cases, of course, no "return case" occurs; but, even so, if a medical man be consulted by anxious parents, the verdict is probably the same.

In many cases, primary desquamation will have ceased

* There were no deaths to exclude.

by the end of a month, but secondary desquamation may set in after the patient reaches home. The following is a case in point. The daughter of well-to-do parents was sent home after being in hospital thirty days, desquamation being apparently completed. She had had a slight attack; and my opinion was then, and still is, that she was fit to be discharged. Thirteen days later, I received (through our matron) the following letter from her mother:—

October 21st, 1901.

DEAR MADAM—My little daughter, E. H., was with you in the hospital, having contracted scarlet fever during a visit to an auntie in Leicester, and she was discharged by you as being free from infection on Oct. 9th. To my great consternation and alarm, she is now peeling on the feet and ankles, the skin coming off in enormous pieces. I *cannot* understand her being sent from a hospital in such a town as Leicester in this condition. Can you explain it to me? Is the skin on the feet any *less* infectious than on any other part of the body? I am in a great state of anxiety, and shall be glad if you will write at once. We would *gladly* have paid any sum rather than she should have come home in this state. I tremble to think what the consequences may be. . . . .

Yours very truly, ———

I wrote to the lady and reassured her. Some time afterwards, I received a second letter from her:—

DEAR SIR,—I am very glad to be able to tell you that no harm came from E.'s second desquamation. She went on peeling for quite four weeks after coming home. When I wrote to you about her being with other children, I did not mean my *own* children. There was only one boy at home, and he had had fever. What I was so alarmed about was my letting her little friends come to spend an afternoon and evening with her. Four little children came on different afternoons, and stayed with her for some hours. They were all younger than E., and none of them had had fever, so that I was much more anxious than if they had been my own. When my housekeeper told me E. had not finished peeling, I did not allow her to be with children again until we found she was quite clear: though, of course, she was going about the house as usual, and going into the fresh air. . . . .

Yours very truly,———

The above case is, I know, merely negative evidence of the non-infectivity of desquamation, but such cases could be multiplied. They are at least a set-off against cases such as those recorded in the Clinical Society's Report, or as the one quoted by Dr. C. B. Ker (*v.* Appendix I), where the only discoverable cause to account for prolonged infectivity is the presence of desquamation. Some of my correspondents make a distinction between primary and secondary desquamation, admitting that they do not regard

the latter as infectious, but apparently are in doubt as regards primary desquamation. I fancy this distinction arises, partly at least, from inability to shake off entirely the old belief in the infectivity of desquamation, and a consequent desire to compromise. I cannot see what scientific reason there is to think that a patient still desquamating, say, at the end of eight weeks, is more infectious if the process has been continuous, than if it has ceased for a time and recommenced. It is interesting to note that in the cases quoted both by Dr. Ker and myself, and also in Cases XXIV and LVIII quoted in the Clinical Society Committee's Report, the desquamation was secondary, and in one case tertiary.

Others of my correspondents seem inclined to fix a time limit to the infectivity of desquamation, regarding it as non-infectious in the later stages. This, again, looks rather like a desire to compromise. In the nature of things, it is scarcely possible at present to prove or disprove the infectivity of desquamation in its early stages, because it almost invariably begins in the first two or three weeks of the disease, whilst a patient is still infectious apart from desquamation. As to where the supporters of a time limit would draw the line, that will evidently depend upon what the individual observer has come to regard as the average duration of infection in scarlet fever cases. In the absence of much fuller and more elaborate statistics than any we yet possess, it cannot be claimed that we have any basis to go upon.

The view that the infectivity of desquamation varies with the stage of the disease practically amounts to this: So long as a patient remains infectious, desquamation is infectious; when he ceases to be infectious, desquamation ceases to be infectious. In other words, desquamation is no criterion of continued infectivity, which is the principal issue I have raised.

There is one other point which I must just mention. I fancy that some are a little afraid, if a disbelief in the infectivity of desquamation should be admitted, of losing a useful popular lever. As things are at present, the public will readily believe a patient is still infectious, and continue to take precautions as to isolation, etc., so long as such a palpable and, as they think, dangerous symptom as desquamation continues. Shake their faith in this, and patients might be running about the streets at the end of ten days or a fortnight! I fully realise the force of this. What Medical Officer of Health, indeed, has not found

it convenient to avail himself of this firmly-established belief? As students of science, however, we ought not to allow such an opportunist consideration as this to obstruct our search after truth. Moreover, our methods and measures for controlling the spread of scarlet fever (based largely upon theories) have not been such a conspicuous success in the past, that we need apprehend any very serious consequences from a little modification of them in the future.

I may, perhaps, be allowed to refer incidentally to my practice of sending cases out as early as I do. It is partly done as a protest against the common practice, futile, as I hold, of trying to isolate all cases for such a period as will cover exceptional cases of prolonged infectivity; in other words, to entirely prevent "return cases." I recognise perfectly that some cases of scarlet fever, even after most careful sorting out of all doubtful cases, will not be free from infection at the end of four or five weeks. But I also recognise that some cases will not be free from infection at the end of eight or nine weeks; no, nor at the end of twelve or fourteen weeks! Why, then, should we fix a minimum period of isolation, as is done in some hospitals, of eight weeks? There is little doubt, I think, that the very great majority of cases, sorting out, of course, special cases, such as rhinorrhœa and otorrhœa, are quite ready to leave hospital in much less time than this. Are we, then, justified in detaining them so long? When we consider that every extra week a patient is detained costs the ratepayers something like 15*s*., and increases the risk to the patient of "catching something" in hospital, whilst the nett gain to the community by this prolonged isolation, in the way of reducing the prevalence of disease, is something so small that we have not yet succeeded in demonstrating it, I doubt very much if we are justified. Certainly, I do think we are justified in trying if we cannot get equally good results whilst reducing the period of isolation.

The scarlet fever at present being met with is a very different disease from what it was only ten or fifteen years ago. In Leicester, during 1901, we only had 6 deaths out of 764 cases of the disease notified. Is it not probable that the great change which has taken place in the type of the disease, in the direction of lessened severity, has been accompanied by a shortening in the duration of infection?

In conclusion: I may briefly sum up the principal

arguments against the supposition that desquamation is infectious, as follows :—

(1) The absence of evidence supporting it. It is difficult to believe but that if the old supposition were correct, strong evidence of it would, ere this, have been forthcoming, as is now the case with discharges from the nose and ears.

(2) The fact that infectivity begins prior to the onset of desquamation, and frequently continues long after desquamation has ceased.

(3) The fact that scarlet fever wards, although abounding in desquamating epithelium, are not a danger to neighbouring houses.

(4) The fact that the proportion of "return cases" does not appear to be increased amongst patients sent out from hospital still desquamating.

On the other hand, the principal argument in favour of the view that desquamation is infectious is the fact that patients still desquamating, but otherwise apparently free from infection, have frequently been known to convey the disease to others. The whole force of this argument disappears, however, as it seems to me, when we consider that just as frequently patients, apparently quite free from infection, and in whom desquamation has entirely ceased, have also been known to convey the disease; moreover, patients still desquamating have frequently mixed freely with others without untoward result.

APPENDIX II.—PARTICULARS OF "RETURN CASES" ARISING OUT OF 190 PATIENTS DISCHARGED AFTER AN AVERAGE ISOLATION OF 39.1 DAYS.

	Infecting Case.			Return Case.		
	Name, Age, and Registered Number of Infecting Case.	Duration of Infecting Case, Dates of Onset and Discharge.	Condition of Infecting Case at Time of Discharge.* and Subsequent Health.	Name, Age, and Registered Number of Return Case.	Date of Onset of Return Case.	Interval between Discharge of Infecting Case and Onset of Return Case.
1	John P., 4 years, 667.	41 days, Aug. 25th to Oct. 5th.	"Looks unhealthy." After return home, suffered from constant nasal discharge.	Albert P., 8 years, 885.	October 15th.	10 days.
2	Ernest G., 4 years, 675.	42 days, Aug. 28th to Oct. 9th.	Crack behind ear not healed.	Ellen G., 2½ years, 886.	October 16th.	7 days.
3	George W., 4 years, 757.	47 days, Sept. 14th to Oct. 31st.	"Nose better, but not quite right. Still desquamating on feet."	Herbert W., 3 years, 985.	November 5th.	5 days.
4	John James K., 11 months, 924.	26 days, Oct. 28th to Nov. 23rd.	"Nose looks suspicious; little discharge."	George K., 7 years, 1046.	November 27th.	4 days.
5	Edward D., 16 years, 929.	39 days, Oct. 26th to Dec. 4th.	"Sore nose." Sore at home after return for more than a fortnight.	Hilda D., 9 years, 1105.	December 17th.	13 days.

* As recorded on bed chart at time patient was "passed."

NOTE.—The Infecting Cases in Nos. 1, 3, and 4, had suffered from Rhinitis whilst in hospital.

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ong	and treatment sequent stages

APPENDIX III.

The following is an example of prolonged secondary desquamation following post-scarlatinal diphtheria :—

Albert P., aged 8 years, Registered No. 885, admitted to the Leicester Fever Hospital on October 18th, 1901, with a slight attack of scarlet fever. Eruption present, November 11th, desquamating on hands ; Nov. 28th, still desquamating on hands, but "passed" to go home on Dec. 4th.

December 3rd. Post-scarlatinal diphtheria developed. Typical patch. D. B. present. No further eruption. Patient detained in hospital.

December 13th. Hands desquamating in sheets.

December 20th. Desquamation on hands ceased. Desquamating freely on feet.

December 30th. Desquamating on both hands and feet. Tonsils enlarged.

January 4th, 1902. Ditto ditto.

January 6th. Ditto ditto.

January 16th. Sides of feet still desquamating freely in flakes. Desquamation on hands ceased. "Passed" to go home.

January 18th. Discharged ; feet still desquamating.

Total time in hospital = 92 days.

Total time from onset of desquamation = 68 days.

N.B.—This patient would have been discharged earlier but for parents' wish to the contrary.

INFANTILE MORTALITY IN THE TROPICS.

By DR. DANIELS,
Medical Superintendent of the London School of Tropical Medicine.

(*Read: March 21st*, 1902.)

THE mortality in infancy and childhood amongst native races in the Tropics is of such importance to the prosperity of our colonies, that I venture to bring a few observations on the subject to your notice.

The accurate statistics to which you are accustomed in England cannot be obtained in most tropical countries, and the fallacies and errors are greater, and in many cases are multiplied by more extensive observations.

The figures I give I consider only as a rough index of the part played by excessive early mortality, in producing the decrease in population which occurs in some parts of the tropical possessions of Great Britain.

General attention in England has not been directed to the subject, though to those who have worked amongst the native races the broad facts are familiar.

The mortality amongst European children is well known to be great, but I do not propose to consider that question here.

In Chart No. I, I have given the rate of mortality for the district of Bengal as the largest in India, and of British Guiana as compared with the mortality at the same periods of life in England, and it will be seen that at every period the rate is greater than in England.

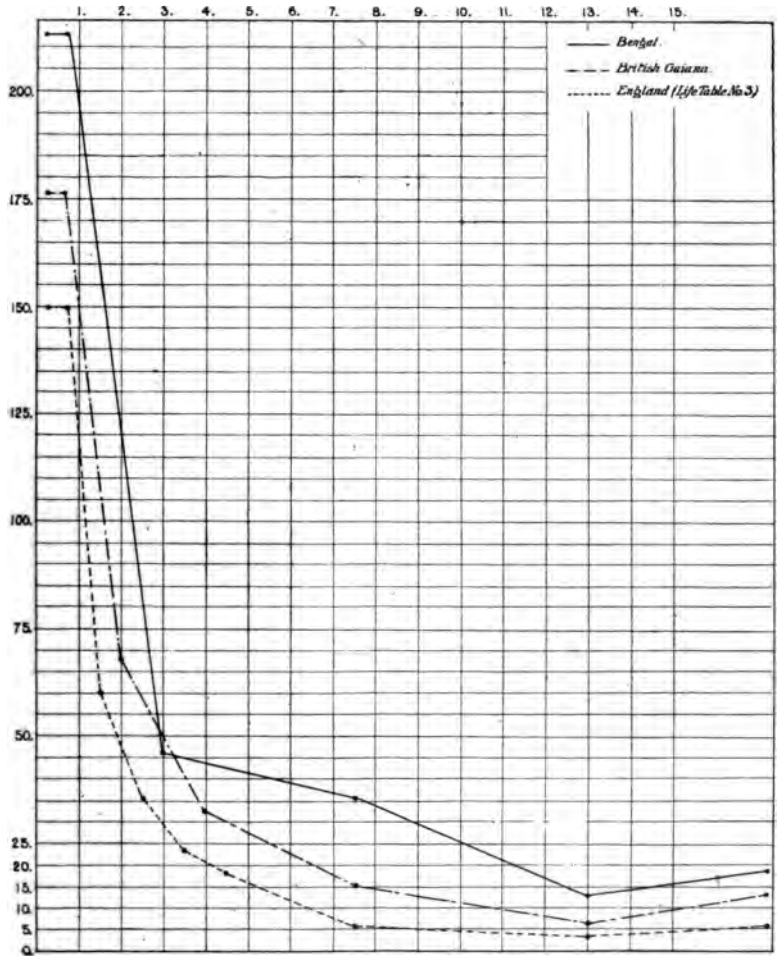
A large part of this mortality is in the first year of life, and though in different districts in India this varies enormously—from 439 in the Central Provinces downwards—still in few districts is it below 200.

In other countries, *e.g.*, Penang in the Malay States, taking one year with another, I am informed by Dr. Kerr that the mortality under one year is usually about one-third of the total births. In Fiji, 35 per cent. of the total deaths occur in children under one year of age; and in both Sierra Leone and Lagos the heavy infantile mortality has attracted the attention of the local authorities.

According to Dr. Prout's sanitary report for Freetown,

for 1897 and 1898, 35.67 of the total deaths occurred in children under 5 years of age; and the mortality under

CHART I.



Comparative Mortality from Birth to 15 in Bengal and British Guiana.

one year was 408.8 and 375.7 for the two years respectively.

In British Guiana the return to Mr. E. A. G. Dalton, the Registrar-General, showed that the mortality under one

year varied considerably amongst the different races there represented, and in 1894 was—for Negroes 205, Europeans 184, and for Indians the low figure of 155. In most years the rates were somewhat higher than these.

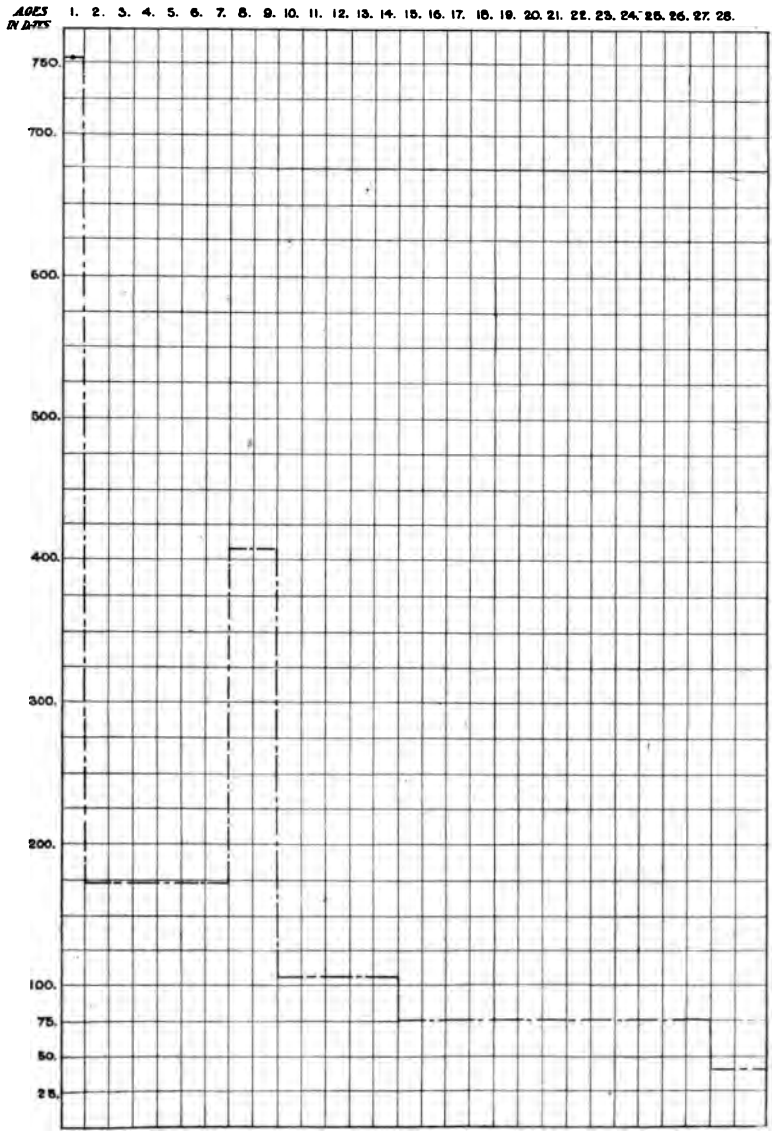
The causes of the heavy infantile mortality are difficult to obtain, as little more than half the deaths were certified by medical men, and even of those certified one-fifth were too vague (debility, etc.) to be of much value.

A further subdivision, showing more in detail the periods at which deaths occurred, gives a further clue to some of the factors in the infantile death-rate. A series of inquiries from 470 negro women showed that they had amongst them borne some 1500 children. The subsequent fate of these children were inquired into, and the results are embodied in Chart II.

2.06 per cent. died on the day of birth, and a further 2.85 in the first week, or 4.91 that could hardly be due to dietetic errors, or to disease acquired after birth; and at the temperature 80-85 deg. F. per cent., exposure and cold could have little or no effect.

These deaths must in the main be due to deficient vitality of the children rather than to external circumstances. They are probably due to similar causes to the still births. This view is supported by the large proportion born dead. In the Georgetown Hospital practice, out of 1383 deliveries, in 20.4 per cent. the children were still-born. As cases of all kinds, normal labours, etc., are admitted, there is no reason why the proportion should be very much in excess of the general average; but even if compared with an English Lying-in Hospital, such as Queen Charlotte's, where half the children born are illegitimate and nearly two-thirds first confinements, the rate is more than four times as great. The certified still births in British Guiana for 1895 were 11.7 per cent. for the whole colony, and 14.3 for Georgetown and district. These figures are probably under the true proportion A heavy early mortality and large proportion of still births are associated elsewhere, as in Fiji, and the cause is most obscure. In Freetown, Sierra Leone, in 1897 and 1898, there were 1,255 births, and 77 or 6.1 were buried in the cemetery as still-born; but, as Dr. Prout points out, it is extremely probable that this does not represent the total number. Of the children born alive 121, or 9.1 per cent., died within twenty-four hours, and 222, or 17.6, within the first week. A marked rise in the mortality in British Guiana is seen on the eighth and ninth

CHART II.



Showing the Death-rate amongst 1500 Negro Children in British Guiana in the First Month.

day, and this is certainly due to *Trismus Neonatorum*, a preventible disease.

From this period onwards there is a steady decline in the mortality, without any marked interruptions, but the rate continues high.

Dietetic errors are doubtless responsible for the greater part of this mortality. A large proportion of the women, married or otherwise, either are engaged in domestic service, field labour, etc. These are unable to nurse their children in the day. Even those working in their own houses feed their children largely on various forms of farinaceous food. Still, the acute forms of diarrhœa which are so common amongst the children of the poor in hot weather in England are very rare in British Guiana. The various diseases of the respiratory organs so common in an English winter are rare in British Guinea. Zymotic diseases are not common, though measles and whooping-cough occur, but are usually very mild. Scarlet fever and small-pox are unknown, and diphtheria is very rare. Malaria, however, is prevalent, and I have seen death result from it as early as the end of the first month ; and after the sixth month it becomes a fairly common cause of dangerous illness or death.

Much attention has in the last few years been paid to the frequency with which native children harbour malaria parasites without obvious signs of illness. The conclusion that has been drawn by some, that this disease is harmless to native children, is, however, erroneous.

Such children are liable to the sudden onset of the pernicious cerebral form, rapidly resulting in convulsions, coma, and death. These attacks are due to the accumulation of the parasites in advanced stages of their growth in the cerebral capillaries. In several of these cases within my own experience, the history, as related by intelligent parents, was as follows: The child seemed all right in the morning, became drowsy in the afternoon, convulsions set in, and the child was brought to the hospital. Energetic treatment with quinine, in the great majority of cases, results in speedy recovery, and if death occurs it is usually within two or three hours of admission. In such cases, post-mortem examination shows the cerebral capillaries to be blocked with corpuscles containing parasites. When minute doses of quinine are given deaths are frequent, or when the nature of the disease is not recognised. Though these attacks affect children of all races, in some families they seem to be more common than in others. I have known

one family of three negro children, within three months, all had such attacks. One died at home, the second was brought to the hospital moribund, and the third was brought as soon as the convulsions commenced, and rapidly recovered.

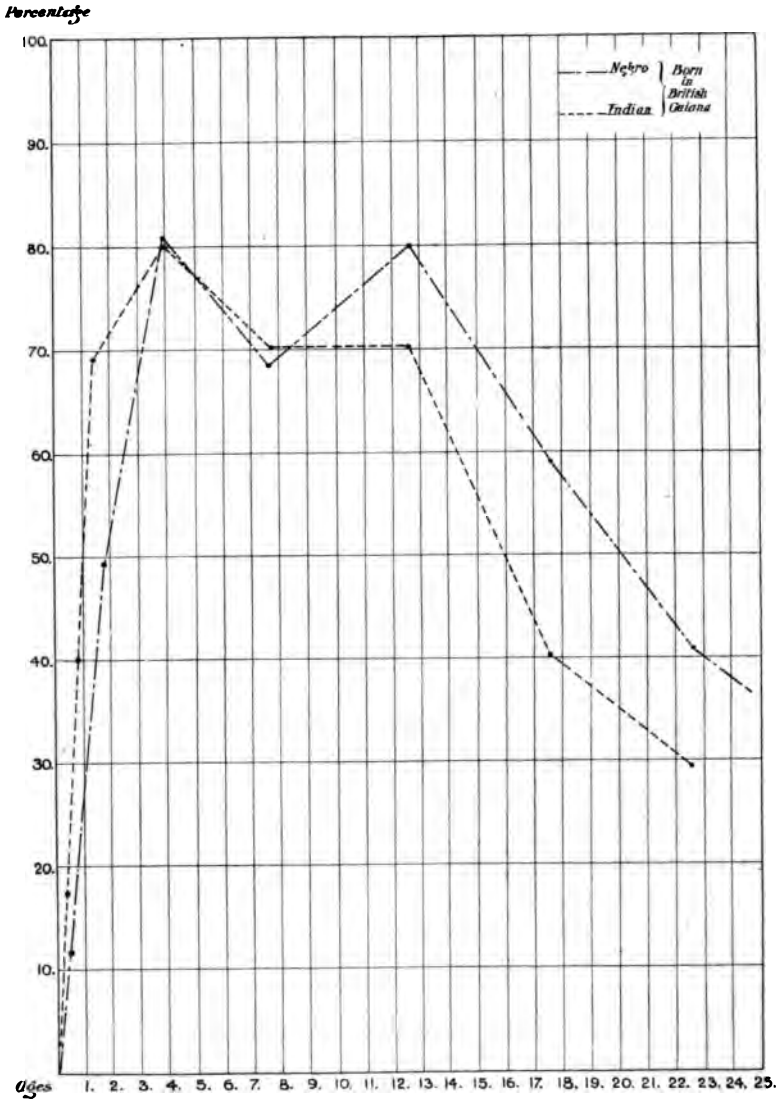
In these children there are none of the classic symptoms of malaria; and unless forewarned, or parasites are found in the blood, a treatment such as is usual for infantile convulsions in England may be adopted, and in the majority of cases death will result.

Of course I do not consider this disease the only cause of convulsions, but only as a common one in early infancy. After six months it is, I believe, the chief cause of such cases of convulsions, and in such as I have examined post-mortem the cerebral condition described has been met with constantly. Convulsions, in great part malarial as I believe, form an undue proportion of the infantile deaths. In British Guiana, out of 1,471 deaths under one year, 158 were due to convulsions, and of the 832 medically certified 79 were attributed to this cause. In addition, 31 of the certified deaths, and 2 of the uncertified, were attributed to malaria. "Simple fever" was responsible for 135 of the uncertified deaths.

Such fatal attacks of malarial convulsions are not found only in the first year of life. They are the common dangerous manifestations of malaria up to about six years of age. In older children, or in negro adults from a non-malarial place, such as Barbados, coma supervenes in malaria without convulsions. It will be noticed in the Charts that in British Guiana the mortality is in decided excess of that in temperate climates all through childhood; and it is difficult to avoid attributing this mainly either to these pernicious attacks of malaria, or to visceral changes resulting from malaria. In India the question is more complicated, on account of cholera, small-pox, and famine. The proportion of children who are infected with malaria, and in the main undergo natural cures, is seen by the percentage of persons with pigmented (malarial) spleens at different ages. These include deaths from all causes, but as malarial cases were usually diagnosed and energetically treated, few of the deaths were caused by acute malaria. Chart III.

The enviable position of Barbados, an island free from malaria but with all other diseases in common with British Guiana, might here be mentioned. In that island there is no proper registration of either births or deaths, but the

CHART III.



Showing the Proportion of Children in whose Spleens Malarial Pigment was found on Microscopical Examination ; 344 examined under twenty-five years of age.

census returns show a steady increase in the population of 6.3 per cent. in ten years; and this does not represent the

total increase, as Barbados has a population of 1,096 to the square mile, and many of the natives emigrate to less crowded colonies. About one-fifth of the negro population of British Guiana are natives of Barbados.

In British Central Africa there are no statistics at all bearing on the subject. Inquiries from native women showed that there also the early mortality is very heavy, and in respect to the mortality in the first week very similar to that of British Guiana, as 2.7 per cent. of 552 children died on the first day, and all through childhood and infancy the mortality was large. The inquiries were made in two districts, one intensely malarial, and there over half the children born died before puberty. The other was less malarial, probably little more so than British Guiana, and there 38 per cent. died before puberty. No information could be obtained as regards still-births. The proportion with malaria is indicated by the percentage of unselected children with markedly enlarged spleens. Chart IV.

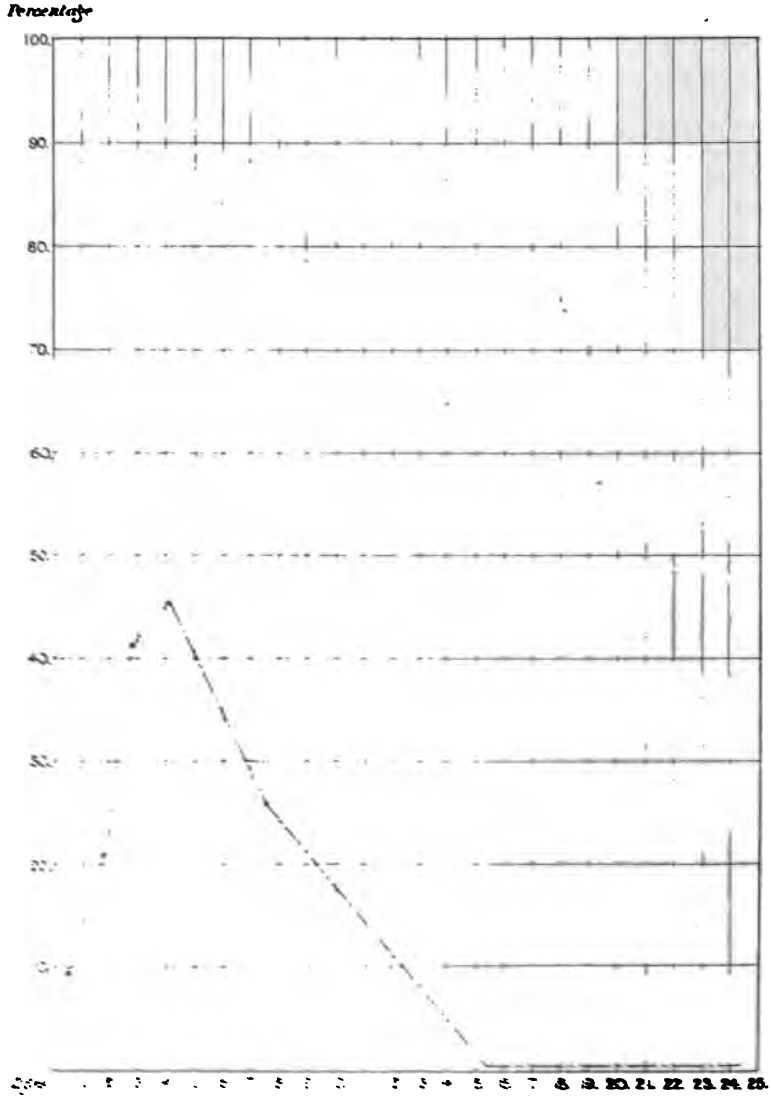
In Central Africa, however, the question is complicated by the existence of small-pox. Epidemics occur every five or six years, and as during each epidemic inoculation is largely practised, the incidence of the disease is heaviest on the children. The case-mortality is stated by those with experience of small-pox, both in England and Central Africa, to be comparatively low.

Enough has, I think, been said to indicate the main outlines of the preventible causes of the mortality. The deaths at or soon after birth are probably due to causes which cannot readily be controlled. Dietetic errors are probably little greater than amongst the city populations in England, and are not likely to be easily remedied.

The disease which can be most certainly diminished is malaria, and a diminution in the incidence of this disease will reduce the mortality in the later months of infancy and childhood, probably to a sufficient extent to result in the increase of the population, instead of, as now, so often occurs a decrease.

It is generally assumed that the natives of tropical countries are very fertile. The evidence that this is usually the case is not conclusive. In India, in the Punjaub, the birth-rate is as high as 43.9 ; but that appears to be exceptional, and in Bengal for the same year was only 32.81, and there the deaths exceeded the births by 2.07 per 1,000. In British Guiana the negro birth-rate varies from 30-35 : not sufficient to replace the loss by deaths,

CHART IV.



[illegible] Africa with markedly [illegible] under fifteen [illegible]

though in some years the diminution in the population is small.

The families in British Guiana are usually small. Inquiries amongst negro women attending out-patient practice, showed that out of 58 fertile women who were past child-bearing age, they had only, including still-births, an average of 6.29, or under 5, if still-births be allowed for.

In British Central Africa 41 fertile negro women, over 45 years of age, also averaged 5 children born alive.

These figures are low, and when the mortality in infancy and childhood is also considered, it is easy to understand why so many fertile tropical countries are sparsely inhabited.

The effect of European administration and organisation has in part been for good, so far as deaths from violence are fewer, and the loss of life consequent on the slave trade has been abolished with that trade.

On the other hand, European methods of cultivation, railway and mining works, tend to aggregation of the natives; and at the same time by interfering with natural drainage or by irrigation, increase the number of *anopheles*, so that the combination of these two factors favours an increase in malaria. Vaccination is imperfectly carried out, or not at all. Other diseases are introduced, and amongst these, I include tuberculosis and syphilis. Even if these diseases are already present, the overcrowding resulting from partial adoption of European methods of housing causes a great increase in their prevalence.

In some of the tropical countries it is unnecessary to assume the existence of any further causes for a decrease in the population. There is sufficient to show that the change in circumstances, and in the resulting prevalence of diseases, is sufficient to so alter the balance of the population, that where before the advent of Europeans the population was stationary, slowly decreasing, or slowly increasing, under the changed conditions there is a rapid decrease.

The recognition of the economical importance of the question is being slowly forced upon us. In few places is the resident native population sufficiently abundant to both grow their own food and labour on European plantations and works.

Labour and food has therefore to be brought from a distance; and now that even in Africa, European influence is so widespread, the demand for labour is more universal,

and can less readily be supplied from other districts, and then only at heavy expense.

To ensure a permanant labour supply in the Tropics an increase in the native population is a necessity. Any other measures can only give temporary relief.

In conclusion, I may state that this paper has had to be prepared earlier than I had intended, and some sources of information I have not been able to consult in the time. The importance of the subject must serve as an apology for this inadequate treatment.

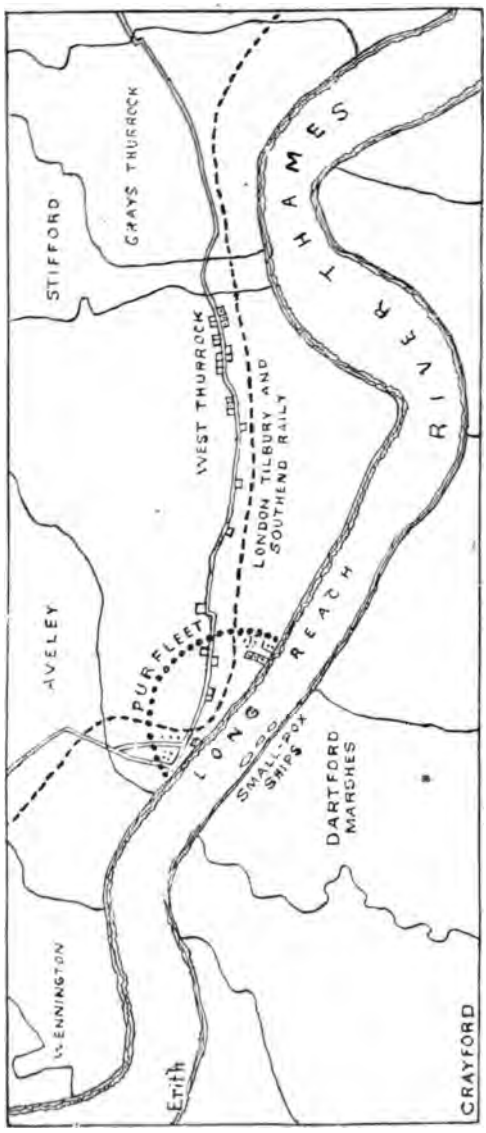
SMALL-POX HOSPITALS AND THE SPREAD OF INFECTION.

By JOHN C. THRESH, M.D., D.Sc., D.P.H.; Lecturer on Public Health, London Hospital Medical College; Medical Officer of Health, Essex County Council.

(*Read: April* 18th, 1902.)

THE prevalence of small-pox in the metropolis, and its consequent spread into the county of Essex, has naturally caused me to give a considerable amount of attention to this disease, in order that I might advise the various authorities in the county how to deal with any cases which might be introduced, promptly and efficiently, to prevent an epidemic occurring. There is a population approaching half a million in the portion of the county abutting upon the east end of London, and as this population consists chiefly of working people residing in somewhat crowded cottages, one would naturally expect that it would be in these densely-populated suburbs that the disease would be most frequently introduced, and in which it would be most likely to become epidemic.

Previous experience in the county, however, had led me to doubt whether this was the most vulnerable point. During the epidemic prevalence from 1893-95, it was observed that the much more thinly populated district included in the Orsett Union suffered far more in proportion than any other part of the county; and my investigation of the cases which occurred there had led me to infer that most of the primary cases had been infected from the small-pox ships of the Metropolitan Asylums Board, which are anchored in the Thames opposite Purfleet. The appended map shows the position of the ships with reference to Purfleet and the Orsett Union. I paid particular attention to these Purfleet cases, making inquiries of the patients and of their friends, and visiting the works and other places where those engaged in work away from their houses were employed. The result left little doubt in my mind that there was some influence at work not common to other districts in which small-pox had been introduced, and



that this factor was the proximity of the hospital ships. Unfortunately, I also found that there was a certain amount of intercommunication between these ships and the Essex

shore, and this afforded a possible explanation of the introduction of the disease, although it was not traceable. It sufficed, however, as an explanation to those who did not believe in the air-borne theory of conveyance of infection; and at the request of the Essex County Council, the Metropolitan Asylums Board undertook to so alter their arrangements that all communication with the Essex shore should be prevented, so far as laid in their power. This fact is of great importance in connection with the present outbreak, as I am convinced that the Asylums Board have fulfilled their promise, and that there has been no personal communication between the ships and the county, save probably the surreptitious visit of a man to his sweetheart. The only case of this kind I can hear of has been denied by the parties concerned.

Dr. John C. McVail, in his Paper on the Aerial Convection of Small-pox from Hospitals, read before this Society during the Session 1893-4, has discussed so fully the possible effect of the removal of patients, movements of medical officers, nurses, and workpeople, on the inhabitants around, that I purpose only referring to these dangers in so far as they have come under my personal observation during the present outbreak in Essex.

(*a*) *Removal of Patients to Hospital.*—Where an isolation hospital is established, one or more persons must be employed in the removal of patients, and the ambulance must pass along streets and highways. We have had complaints of the men in charge of such vehicles stopping at public-houses, and of groups of children standing round the ambulances, their curiosity being aroused by the peculiarities of the vehicles. I have not, however, yet met with a case which could be attributed to infection from this vehicle; and along the routes to two of the hospitals in this county which have received hundreds of cases, no indications have been found of the disease being spread in this manner, though I am not prepared to say that such is impossible. The driver of one ambulance, who unfortunately was not re-vaccinated, contracted the disease, although he did not touch either the patient or any article removed in the ambulance. The danger arising from the removal of patients, providing every care is taken, is, in my opinion, infinitesimal.

(*b*) *Visits of Friends.*—That persons visiting relatives or friends in a small-pox hospital often contract the disease is a well-known fact, and has led the hospital authorities to interdict all such communication save when a patient is *in*

extremis. Unfortunately, visitors frequently refuse the offer of re-vaccination, and no authority has proved strong enough to make re-vaccination imperative before permitting a visit. Cases arising from this cause have occurred in connection with two hospitals in the county, and several persons who have visited at the hospital ships have been attacked. Amongst the poorer classes the dread of infection is not marked, and, as a consequence, they often go to the hospitals either out of mere curiosity, or on the chance of seeing some friend. At the large hospital at Dagenham, to be referred to later, so many people come on the Sundays and attempt to climb the fences to see or communicate with the convalescents, that special police, and all the attendants who can be spared, have to be set on to patrol the ground outside the hospital boundary. Obviously such visitors run some risk, but I have only heard of one case where it appeared probable that this fence-climbing had been the source of infection.

Intercourse of Staff with Persons Outside.—No case has come under notice of infection being conveyed outside by medical attendants, nurses, or other members of the staff of any hospital in the county; but one case has come to my knowledge where small-pox occurred in a house to which a nurse had sent a cloak. Where proper precautions are observed this danger is obviously not great, and in my opinion it has been greatly exaggerated by persons opposed to the air-borne theory, and by medical men and others who have little or no experience in connection with small-pox hospitals. In my opinion it offers no explanation of the prevalence of the disease in the proximity of a well-managed hospital.

Workmen Employed near a Small-pox Hospital.—It is practically impossible to provide a permanent hospital sufficiently large to cope with any outbreak which may occur: hence in many cases temporarily buildings have to be erected in proximity to the permanent hospital, whilst the latter is full, possibly to overflowing, with acute and convalescent patients. Amongst persons so employed infection is very frequent, and occasionally such workmen have introduced the disease into their families, although they themselves have not been attacked; that is, they have been the intermediaries for the conveyance of the infection. Amongst the men employed in putting up temporary buildings at the Orsett Hospital, 5 were attacked with small-pox, and the brother of a man working there was attacked. Up to the present I have only heard of 2 cases

amongst the men erecting the buildings at the hospital at Dagenham, owned by the Corporation of West Ham, and used also by arrangement by several adjoining districts in Essex; but as most of the men reside outside the administrative county, I have not yet been able to ascertain how many have been attacked. In East Ham and Ilford I have reports of 6 persons notified, who had been engaged on the temporary buildings now being erected by the Metropolitan Asylums Board, and of 2 other cases apparently infected by relatives working at the same place. In one instance a man infected his brother, and in the other the son infected his father. By the courtesy of Dr. Richmond, I have been able to examine his books giving the results of his investigations of the origin of the cases in the Dartford Rural District, in which the new hospitals of the Metropolitan Asylums Board are situated, and out of 115 persons attacked no less than 24 were employed at the hospitals. In the urban district of Dartford, Dr. Hamilton informs me that there have been 221 cases of small-pox since September 20, 1901, when the first occurred. Of these no less than 95 were employed at the hospital buildings, 41 were infected by workmen employed at the hospital and residing in the same house as the persons attacked. Out of the remaining 85 cases only 5 could be fairly definitely traced to a pre-existing case or probable source of infection. Several metropolitan borough medical officers of health have informed me that cases have arisen in their districts amongst workmen employed at the temporary hospitals, and amongst their families. The total number of the cases due directly and indirectly to this cause, we may never know; but, certain it is, that these men are spreading the disease broadcast through the districts to the East of London.

It is obvious, therefore, that a considerable amount of danger is attached to working in close proximity to small-pox hospitals where a large number of patients are under treatment, and the question naturally arises, how do these people become infected? I have not heard of or observed any similar results ensuing amongst men engaged erecting temporary hospitals for other diseases; and from our knowledge of such diseases as scarlet fever, diphtheria, and typhoid fever, we should not expect to find infection spread in this manner. Doubtless direct infection is responsible for a certain number of cases; but it appears certain that most of the infected persons have never come in contact with any patient, and that some other explanation is necessary to account for the large number of cases

which occur. This factor is the conveyance through the air of the infective material from the patients in the hospital to the persons around. That infection can take place without actual personal contact is generally, if not universally, admitted; but whilst some would limit the "striking" distance to a few feet, others will extend it to hundreds of yards. This brings me to the most important part of my subject: the aerial conveyance of infection from small-pox hospitals.

Aerial Conveyance of Infection.—In 1894, Dr. McVail, in the Paper already referred to, stated that his purpose was "rather to review existing knowledge than to adduce new facts;" and in his most admirable and instructive review he shows that this is no new theory, but one which has been frequently discussed during the last hundred years. In fact, wherever there has been a large aggregation of persons suffering from small-pox, cases appear to have occurred amongst the inhabitants around, which could only be accounted for by the infection being air-borne. After considering the arguments for and against aerial convection, he says:—

"Summing up the evidence, as far as it is possible to sum up on a subject as to which so much is still to be learned, we seem to reach the general conclusion that, as a result of the simultaneous action of causes favourable to the spread of infection, the contagium of small-pox may be conveyed atmospherically to a distance much greater than had been usually admitted—a distance measurable by quarters of miles." He also adds: "The practical conclusion of this whole question may be said to have already been arrived at. Small-pox hospitals are not now erected in the midst of towns, and those already in existence are being more and more sparingly used. Indeed, when the power of aerial convection is still doubted, it seems to be assumed that the prevention of personal communication is impracticable, and that accidents incident to the system of hospital treatment of small-pox within populous districts must be accepted as inevitable; so that the only remedy under the one theory, as under the other, is the removal of such institutions to a distance from populous places."

I differ from Dr. McVail, however, in not believing that the practical conclusion has been arrived at. It is probably not sufficient in all cases to place such institutions at a distance from populous places. It may be necessary to avoid having large institutions, with consequent large concentration of patients, and therefore of infective material, by

substituting for these a number of smaller hospitals, or a number of tent encampments, in which this concentration of infection cannot occur, or only to such an extent as to reduce the danger of the dissemination of the disease to a minimum. Small-pox hospitals do not necessarily cause widespread prevalence amongst the population around; on the other hand, they may spread the disease over a greater area than is generally conceived. The experience in Essex during the present epidemic has been particularly interesting, and will probably tend to throw a little more light on some points of this much-discussed question of aerial conveyance of infection. During this and previous epidemics I have seen a number of cases isolated in their own homes without any spread of infection, either among the inmates of the respective houses, or amongst the neighbours where the house has been one of a row or group. In most of these cases revaccination has been submitted to by the other inmates of the houses, not always by all the inmates, and generally by very few of the neighbours. My experience has not led me to recommend the County Council to urge the provision of a few large hospitals to serve the whole county, but rather to urge each district to provide temporary accommodation for such cases as may occur, and I always recommend the use of tents. Throughout this epidemic I have had charge of three such hospitals, and I have on previous occasions had charge of similar ones in which from 1 to 8 cases have been treated at a time, and there has never been the slightest reason to believe that any person has been infected from such hospitals, although they have sometimes been near groups of houses or populous villages. With large hospitals my experience has been somewhat different. To take first the Orsett Hospital, which serves the Orsett Union, comprising the Orsett Rural District and Grays Urban District, this hospital was originally erected for small-pox, in consequence of the prevalence of this disease in the district during the epidemic of 1884, when the cases from London were first sent down in any number to the hospital ships in the Thames. After this epidemic had ceased, it remained unused so long that the authorities decided to use it for infectious diseases generally, and it was being so used when small-pox broke out in London last year. Directly cases began to occur in Purfleet, the patients in the hospital were removed, and the small-pox cases taken in. About twenty beds were available, but cases cropped up so rapidly that additional accommodation had to be provided in the shape of wood

and iron buildings and hospital tents. The maximum number of patients in the hospital at one time was over 100, and they certainly were crowded together more than was desirable. Several workmen engaged on the temporary buildings were attacked with the disease, but only one other person working or living near has been infected, so far as I can learn. The hospital, however, is on fairly high ground, and very isolated. There appears to be only one house within a quarter of a mile, and only three or four between a quarter and half a mile, the same number between half and three-quarters of a mile, and only one small group of houses just within the mile. Beyond this limit lies the town of Grays, nearly the whole of which lies within the mile and a half radius. The distribution of small-pox in the parishes around does not appear to indicate that this hospital has had anything to do with the aerial dissemination of infection, but it is just possible that the influence, if any, is masked by the effect of other and more important factors.

The small-pox hospital at Dagenham is much more important than that at Orsett. It is a properly-equipped hospital, with sixty beds and an administrative block sufficiently large to serve a hospital three or four times the size. It was provided to serve a population of over half a million. It stands in the centre of an estate of 120 acres, and though not so isolated as the Orsett hospital, the nearest village, Dagenham, is beyond the half-mile radius. The spread of small-pox in West Ham and other districts connected with this hospital has recently been so rapid that temporary buildings have had to be run up as quickly as possible. At my last visit (April 2nd), there were 350 patients and 40 more were waiting for admission. As I have already remarked, a number of persons working here have been infected, and there is considerable communication with the village of Dagenham, in which is the nearest railway station. Up to the present there has been no epidemic prevalence in this parish, but 2 cases occurred there in January, 2 in February, and 7 in March; and it is very probable that the influence of the hospital is beginning to make itself felt. The progress of events is being carefully watched, and the results will be fully noted.* Already the proportion of cases to population is higher in Dagenham than in any other town or village in the Romford Union, save that of Rainham, but as yet it would be unsafe to draw any inferences.

* The disease has since spread into the adjoining parish of Hornchurch, where many cases have occurred during the present month (April).

The hospital ships of the Metropolitan Asylums Board, with reference to which I have already contributed an article to the *Lancet*, which appeared in the issue of February 22nd of this year, lie in the Thames just outside the county. They were placed opposite Purfleet in 1884, and since that time there has been an excessive prevalence of small-pox in the Orsett district, and especially in that portion lying nearest the ships. The deaths from small-pox in the Orsett Union and in the remainder of the administrative county of Essex, are given in the subjoined Table; divided into two periods, the first for thirteen years prior to the ships being used for isolation purposes, and the second for the eighteen years after that date.

—	Deaths from Small-pox.		
	In Orsett Union.	In Remainder of County.	In London per Million Population.
1871-80	24	350	457
81	0	18	619
82	1	16	111
83	0	5	35
	25	389	
1884	9	20	313
85	14	49	357
86	0	6	6
87	0	1	2
88	0	0	2
89	0	0	—
1890	0	1	1
91	0	3	2
92	0	2	10
93	4	3	48
94	1	27	20
95	5	4	13
96	0	0	2
97	0	0	4
98	0	0	0
99	0	0	1
1900	0	0	1
1901	16	11	5
	49	127	

Mean population, first period	16,456	337,612	
Death-rate per annum per 1000 population	.117	.089	.41
Mean population, second period	27,000	480,000	
Death-rate per annum per 1000 population	.101	.015	.044

Prior to the floating hospitals being established near Purfleet, small-pox was little more prevalent in the Orsett district than in the remainder of the county, and only from one-third to one-fourth as prevalent as in the metropolis. The change since then has been most marked, for on the same basis the disease has been seven times more prevalent in Orsett than in the remainder of the county, and two-and-a-half times more prevalent than in London. If the decrease in the Orsett district had been as great as in the remainder of the county, the death-rate would have been .019 instead of .101, or less than one-fifth; whereas, had the fall been as great as in London, the death-rate would have been .0125, or less than one-eighth the observed rate. In other words, small-pox is from five to eight times more prevalent in Orsett than it should be, taking the number of deaths as the basis of our calculation.

In investigating the cause of this excessive prevalence it is necessary to ascertain the particular parishes in which the disease has occurred. Unfortunately, I have no personal knowledge relating to the epidemic of 1884-85, but the statistics for the years 1893, 1894, and 1895 are particularly interesting and instructive. They are given in the following Table:—

—	Cases in West Thurrock.	Cases in Remainder of Ossett Rural District.
1893 ...	21	... 32
1894 ...	7	... 7
1895 ...	11	... 7
	39	46

Out of the 85 cases which occurred in the whole of the Orsett Union, with a population of over 27,000, no less than 39 occurred in the parish opposite the small-pox ship with a population of only 2,540. That is, the disease was nine times more prevalent in West Thurrock than in the remainder of the Union. Comparing West Thurrock parish only with the other rural portions of the Union, the excessive prevalence is far more marked. Out of a population of 12,500 in these parishes there were only 8 cases, whereas, as we have seen, there were 39 cases amongst the population of 2,540 in West Thurrock, the proportion being 26 times greater in the parish opposite the ships. That the proximity of the ships was in some way the cause of this excessive prevalence I was convinced at the time of the outbreak; but, inasmuch as there was a certain amount of intercommunication between the ships and West Thurrock, there was left

room for doubt as to the mode in which the contagion was conveyed: a doubt which was taken advantage of by those who did not believe in the theory of aerial convection. As this intercommunication has been entirely cut off, the outbreak of small-pox in the Orsett Union, which commenced in September 1901, is well worthy of study. Many of the factors which have complicated the investigations of the prevalence of small-pox in the neighbourhood of other hospitals are practically absent, and the study is greatly simplified.

At the end of August the number of cases in the small-pox ships was 72, a greater number than had been under treatment at one time since the epidemic of 1894. The first case in the Orsett district was notified on or about this date, and the patient worked on the railway at Purfleet, at the nearest point to the hospital ships, but resided in Grays. The notifications since that date have been as under :—

—	Grays Urban.	Orsett Rural.	Total for Orsett Union.	Total for Remainder of County.
1901—				
August .	1	0	1	0
September .	2	8	10	2
October .	0	9	9	5
November .	3	33	36	24
December .	32	73	105	34
1902—				
January .	55	74	129	99
February .	35	59	94	94
March .	29	30	59	206
Totals .	157	286	443	464
Population, 1901 Census	13,831	19,890	33,721	782,803

In the eight months during which the disease has been prevalent in the county, the attack-rate per 1,000 inhabitants has been as under:—

			Ratio.
In Grays Urban District	...	11.4	... 19
In the Orsett Rural District	...	14.4	... 24
In the Orsett Union ...	...	13.1	... 22
In the remainder of the county	...	.6	... 1

The ratio shows that small-pox during this epidemic, up to the end of March, has been 22 times more prevalent in the Orsett Union than in the remainder of the county, and that the incidence has been greater in the thinly-populated

rural portion than in the urban portion of that Union. Until last month (March), the disease did not show any tendency to become epidemic in any other district than Orsett, notwithstanding that it was being almost daily introduced from London, Dartford, and elsewhere, into the teeming population adjoining West Ham and the east and north-east of London.

Having demonstrated that the Orsett Union was the first part of the county to be invaded, and the only part as yet which has suffered from any epidemic prevalence, the distribution of the disease in the various parts of that Union may be considered. The district comprises eighteen parishes, the position of each relative to the hospital ships being shown on the map (p. 102).

The population and number of small-pox notifications from August, 1901, to April 1902, both inclusive, are given in the subjoined Table.

	Population.	No. of Cases Notified.
Aveley	1061	28
Bulphan	279	—
Chadwell St. Mary ...	4762	0
Corringham	817	4
East and West Tilbury ...	924	49
Fobbing	412	0
Horndon-on-the-Hill ...	568	0
Laindon Hills	233	0
Little Thurrock ...	1198	7
Mucking	464	0
North Ockendon ...	339	0
Orsett	1326	11
South Ockendon ...	1059	31
Stanford-le-Hope ...	1750	13
Stifford	1075	21
West Thurrock ...	2585	118
„ Garrison ...	215	0
„ Reformatory Ship ...	266	0
	19,332	281
Grays Urban	13,831	157
	33,163	438*

The most striking point in connection with this Table is that no less than 117 out of the 438 cases occurred in one parish (West Thurrock), and that that parish is the one nearest the hospital ships. This gives 117 cases amongst a

* The total number of cases, as notified to me monthly by the Medical Officers of Health, is 443, as given in the previous Table. This does not quite correspond with the detailed list with addresses furnished weekly, but the slight error is immaterial.

population of 2,585 against 321 in a population of 30,578. The attack rate per 1,000 population has been as under:

In West Thurrock	...	...	45
In the remainder of Orsett Union	...	...	10.5

or 4½ times higher in West Thurrock than in the remainder of the district.

Most of the cases at Tilbury are directly traceable to personal infection, and the outbreak is one of the ordinary character. The patients are either dock labourers or members of their families, and the infection once being introduced into their crowded dwellings, the extension of the disease presents nothing remarkable.

West Thurrock, however, has a working-class population of a better character. It is a large parish, its area being 2,898 acres, giving more than 1 acre to each inhabitant, and from east to west extends for a distance of 3 miles. It is necessary, therefore, to further consider the distribution of the disease within the parish. The hospital ships are situated 700 yards from the nearest portion of the parish, and right opposite them is a small hamlet called Purfleet. This hamlet is exactly included in the three-quarter mile radius from the ships. . Between this and the two-mile radius the population is very scattered and few in number, but just beyond this radius lies the village of West Thurrock.

The hamlet of Purfleet comprises 110 houses, chiefly cottages, and the population, according to a census taken by Mr. G. Sowden, M.R.C.S., my assistant, is 479. In Purfleet there have been 47 cases of small-pox, and in the remainder of West Thurrock (chiefly in the village 2 miles away) 70 cases. The attack-rate in the two portions is therefore as under:

In Purfleet hamlet	...	100 per 1,000 population
In West Thurrock village	...	33 „ „

In other words, small-pox has been three times more prevalent in Purfleet than in the remainder of the district.

Just within and on the border of this three-mile radius lies also the populous portions of the parishes of Aveley and Stifford. In these parishes (about equally affected) have been 49 cases amongst a population of 2,136, giving an attack-rate of 23 per 1,000.

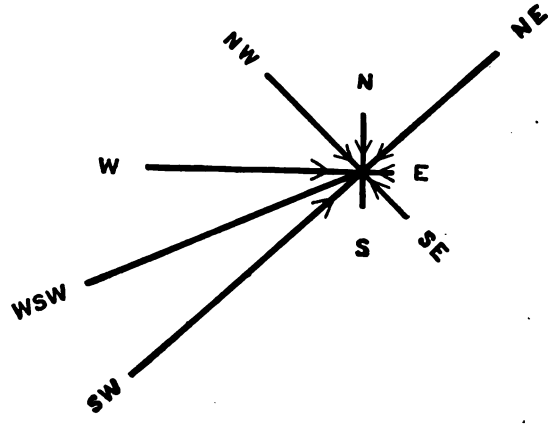
Between the third and fourth mile radius lies the town of Grays and village of South Ockenden. These together have a population of 14,890, and amongst this population there have been 188 cases of small-pox, equal to an attack-

rate of 12½ per 1,000. Beyond this four-mile limit lies the remainder of the population of the Orsett Union, numbering 13,552, and amongst these there have only been 84 cases, equivalent to 6 per 1,000 population.

Summarized, the results obtained are as follows:

Attack-rate per 1,000 Population:	
Between the 700 yards and 1¾ mile radius ...	100
Between the ¾ mile and 3 mile radius—	
In West Thurrock	33.
In Aveley and Stifford	23.
Between the 3 mile and 4 mile radius	19.5
Beyond the 4 mile radius	6.

Strong as are these proofs of the effect of the proximity of the hospital ships, they can be further strengthened by a consideration of the effect of the prevalent winds. The wind rose appended shows that by far the most prevalent



winds during the past eight months have been from south-west and west-south-west, blowing from the ships over the south portion of Purfleet hamlet; hence, if the infection is carried by the air, we should expect to find that the great majority of the cases of small-pox would have been in South Purfleet. This is exactly what has occurred. West Purfleet has almost entirely escaped, whilst South Purfleet has been more than decimated. The two portions of this hamlet are entirely esparate, the one lying to the west of the London, Tilbury, and Southend Railway, and the other to the south, the railway describing a curve equivalent to a right angle between the two parts, *vide* map. There is an interval of about quarter of a mile

between West and South Purfleet, upon which there are very few houses.

In West Purfleet, with a population of 137, there have been only 2 cases; whereas in South Purfleet, with a population of 342, there have been 45 cases.

Attack-rate in South Purfleet ...	...	131 per 1,000 !
West Purfleet ...	...	15 „

The statistical evidence in favour of there being some central focus of infection appears quite conclusive, and confirms the results of inquiries made by myself and assistants in the district concerned. I am convinced that there is no other factor than the presence of the hospital ships which can explain the above facts. The majority of the cases have occurred in the rural portion of the district, and the steps taken to prevent extension were fully as prompt and adequate as in other districts. That a considerable number of cases should occur in the block dwellings at Tilbury amongst the dock labourers, when once the disease had been introduced, is not a matter for surprise, but that there should be a continuous crop of cases—which has extended now over a period of eight months—in a small hamlet like Purfleet, and in spite of all the efforts of the sanitary authority advised by myself, and receiving the special attention of the Local Government Board, alone proves that there is some continuous focus of infection near, and this focus can only be the hospital ships of the Metropolitan Asylums Board.

It has been urged that the magnitude of the epidemic in the Orsett Union is not, save in a remote sense, to be attributed to the influence of the hospital ships. The extensive prevalence of small-pox which has occurred in Grays Urban District, in the village of Orsett, in Tilbury and in other parts of the Union some miles away from the ships, is said "rather to be referred, for example, to spread of the disease within households, or from person to person, or by unrecognised cases of small-pox; in other words, the causes which (as now in London) operate to maintain or multiply small-pox under circumstances in no way peculiar to the Orsett Union. And there can be no doubt that such agencies have also played a not-inconsiderable part in portions of the Union which may be regarded as possibly or probably exposed to opportunities of air-borne infection from the ships, for instance, Purfleet and West Thurrock."* The writer quoted can scarcely

* From a Memorandum by Dr. Buchanan, Local Government Board, March 19th, 1902.

have fully informed himself of all the facts of the case as above recorded, otherwise it is inconceivable how he could have arrived at such a conclusion. No one denies that all the usual factors are operative; but why, if there is no unusual factor, have all the usual and most modern methods of arresting the spread of the disease utterly failed, even when applied in this thinly-populated district? How can the peculiar incidence of the disease—not only in this but in the preceding epidemic—be explained if the factor of the presence of the hospital ships is not of primary importance? I do not for a moment contend that all the persons attacked have been directly infected from the hospital ships. A large number of cases could be traced to direct personal infection, especially in the portions of the district more remote from Purfleet; but many of these could be ultimately traced to Purfleet. Again, it has been urged that the fact that no cases have occurred amongst the garrison at West Purfleet, or amongst the lads on board the training ship *Cornwall*, both within the ¾-mile radius of the ships, prove that the infection is not air-borne. Certainly, at first sight, it does appear singular that these should have escaped, but the reason is very simple. The barracks lie to the extreme north of Purfleet, in the portion where only 2 cases of small-pox have occurred; but the immunity of the garrison is due, not so much to the position of the barracks, as to the fact that every inmate has been re-vaccinated, save a few who had had small-pox previous to the present outbreak. Until all had been re-vaccinated, residents inside the garrison were restricted in their communication with affected areas, but such restriction has now been removed. Major Angell informs me that there are now 224 persons resident inside the garrison. These persons are, therefore, all immunised, and had any cases occurred our surprise would have been great. On board the training ship there are 270 persons, all over 12 years of age. With reference to these, Rear-Admiral Morrell, the Superintendent, writes me as follows: "We go in strictly for vaccination and re-vaccination; but with regard to the staff I have not insisted on it, as I have no power to do this. But many of them have been re-vaccinated, and as most of my men are navy men, they have been re-vaccinated in the service, and my servants and family have all been re-vaccinated." Here, again, we have a community protected by re-vaccination, but probably not so completely protected as the garrison, as cases of small-pox have

occurred on board this vessel which could not be definitely accounted for. In one case a lad, who had not been off the ship for eight months, was attacked, and in the other case the patient was one of the staff.

The escape of the persons residing in the garrison and training ship is a very powerful argument in favour of re-vaccination, but has no bearing whatever upon the question of air-borne infection from the small-pox ships. Another attempted explanation of the excessive prevalence of small-pox in the Orsett Union is the laxity shown by the guardians in recent years in enforcing vaccination. Assuming this to be the case, it does not afford any explanation of the continued prevalence of the disease or of its peculiar distribution: a peculiarity which it must be remembered is common to the previous epidemic. This laxity on the part of the guardians has only been marked during recent years; and in the following Table, compiled from the reports of the Medical Officer to the Local Government Board, it will be found that this argument has very little weight. For comparative purposes I have included two other districts, because in character of population, etc., they resemble Orsett.

Number of Unvaccinated and Postponed Vaccinations per 1,000 *Births.*

—	1883-1887.	1887-1892.	1893.	1894.	1895.	1896.	1897.
Orsett	7.3	13.2	22.1	30.9	42.5	54.7	20.9
Rochford ...	4.1	8.7	19.8	21.5	24.5	30.8	35.7
Romford ...	6.0	14.6	13.9	20.3	19.8	22.6	24.8
County of Essex	—	11.7	16.8	18.5	18.9	21.3	25.8

The figures for the period 1898-1901 are not available. The above statistics show that for a few years vaccination was greatly neglected; and its bearing upon the present epidemic is shown by there being a preponderance of cases amongst children under 10 years of age, and by an unusually high proportion of cases being unvaccinated. This is actually the case, as is shown in the following Table:—

Persons Attacked with Small-pox.

	Under 10 Yrs. of Age.	10 Years and Over.	Total.	Percentage under 10.
Grays Urban	33	.. 130	... 163	... 20
Orsett Rural	58	... 218	... 276	... 21
Other districts in Essex ...	60	... 315	... 375	... 16

Attacks amongst Children under 10 *Years of Age.*

	Vaccinated.	Unvaccinated.	Percentage Unvaccinated.
Grays Urban... ...	1	28	96½
Orsett Rural	7	28	80
East Ham and Walthamstow	20	35	63½

My statistics with reference to vaccination are very incomplete, but those included in the above table are, I think, reliable. In any case they demonstrate the importance of vaccination, and show that the susceptible portion of the population are most likely to be attacked even when the contagium is air-borne, which no one will be inclined to dispute. They also show that the general population in the Orsett Union was not adequately protected by vaccination; and it would be idle to deny that this fact would tend to increase the difficulty in stamping out the disease, but it is utterly inadequate to explain all the facts recited. The next Table, compiled from information obtained by Dr. Sowden when making a census for me at Purfleet, proves, however, that whatever may have been the condition of Orsett Union generally, the people living at Purfleet were probably as well vaccinated as in any hamlet in the county.

Statistics of Vaccination in Purfleet.

—	Vaccinated in Infancy only.	Vaccinated and since Re-vaccinated.	Not Vaccinated, but had Small-pox.	Neither Vaccinated nor had Small-pox.	Total.
Children Under 12 Years of age	70	106	2	7	185
Adults	110	171	8	4	294

There is nothing in this Table to account for 10 per cent. of the population having been attacked with small-pox during the past eight months.

As the result of my investigations, the conclusions arrived at are as follows :—1. That amongst the cases which occurred in Purfleet, both in the present and preceding epidemic, there were a large proportion which could not be traced to pre-existing cases. 2. That all the usual factors tending to produce epidemic prevalence of small-pox were present in the Orsett Union, with the addition of (save in the case of Purfleet) an unusually large proportion of unvaccinated children under 10 years of age. 3. That none

of these usual factors are capable of explaining the peculiar distribution of the disease during the epidemics investigated. 4. That all the results point to some central continuous focus of infection corresponding exactly in position with the small-pox ships. 5. That most careful inquiry fails to show any means whereby the continuous flow of infection can occur except on the hypothesis that it is air-borne.

Many important matters arise for discussion if the theory of the aerial convection of small-pox from hospitals is accepted, but time will not permit of my dwelling upon these at any length. Probably the most important is the question whether the danger arising from the proximity of a small-pox hospital can be avoided, and fortunately my experience in the Orsett district enables me to answer this in the affirmative. If every child under 10 has been vaccinated, and every person over that age re-vaccinated, there is practically nothing to fear. This is proved by the experience of the persons—over 500 in number—residing in the training ships and in the barracks at Purfleet, within the area in which the infection has been most intense. Amongst the inhabitants of South Purfleet only one re-vaccinated person has been attacked, and it is quite possible that in this case the re-vaccination, done five years previously, had not been satisfactory. Amongst the children under ten years of age in Purfleet, 6 have had small-pox, but of these 2 only had been vaccinated and 1 of these imperfectly, as only two faint and small marks could be discerned.

The extent of the area around a small-pox hospital which may be affected directly and indirectly by the hospital is apparently much larger than has hitherto been supposed. In the case we have been considering, the influence is probably being felt at a distance of fully three miles, and the presence of a belt of water half a mile in width is powerless to arrest the spread of the contagion.

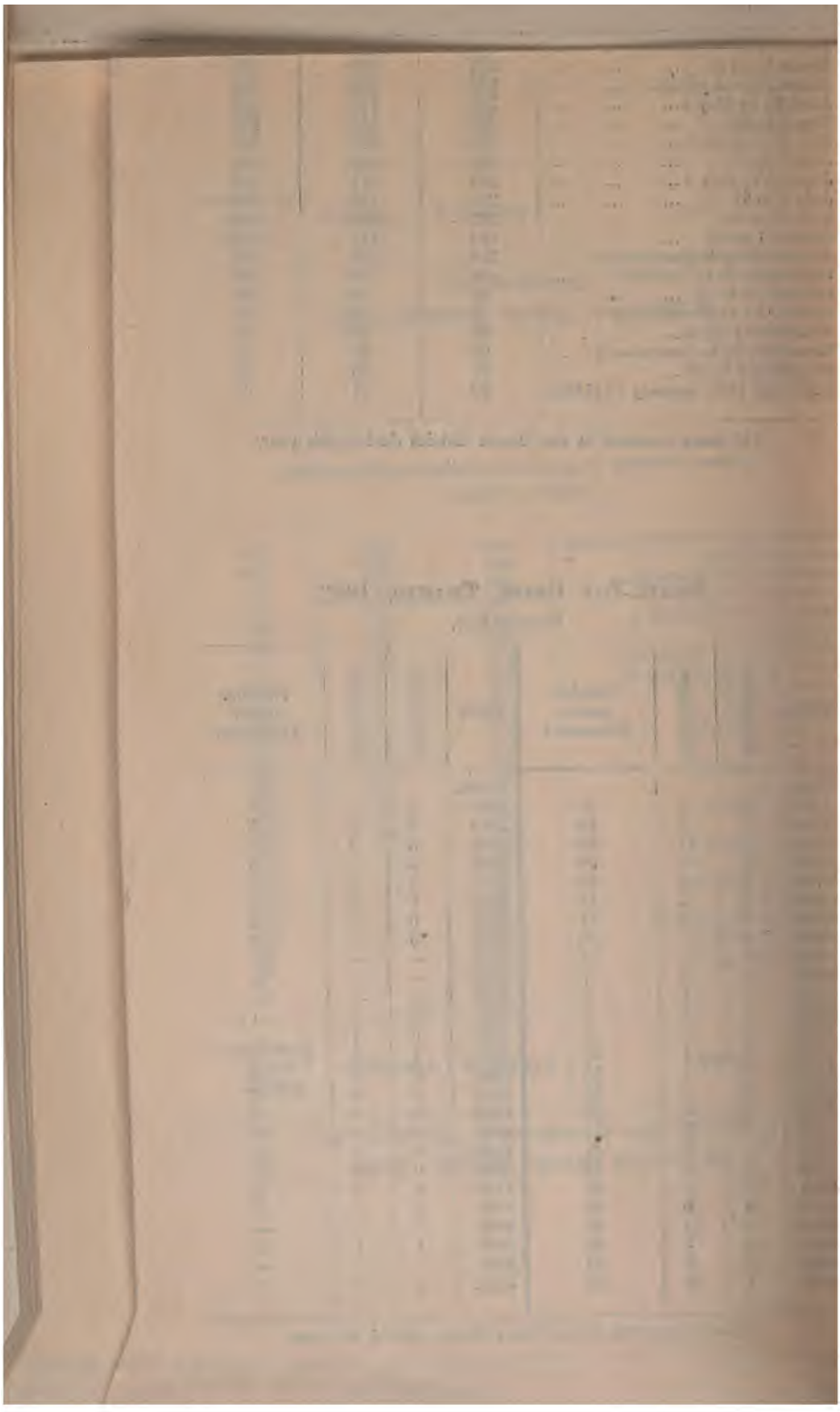
There can be no doubt that the danger increases with the proximity to the hospital and with the increase of the number of acute cases under treatment, the infectivity not being marked until a certain degree of concentration is reached.

With a small hospital, say one constructed for from 10 to 20 or 30 cases, my impression is that there is but little danger of the disease being spread therefrom, providing the site is such as corresponds to the requirements of the Local Government Board; but the danger cannot be

said to be non-existent. With hospitals having a hundred or more beds the danger is naturally much greater, and when we come to hospitals of the size of those required to cope with an epidemic in a large city the peril may be great indeed.

If the Government would learn the lesson taught by this epidemic, and render absolutely compulsory the vaccination of all infants and the re-vaccination of all children between the ages of 10 and 12 years, small-pox would speedily disappear, and hospitals for the isolation of patients suffering from the disease would no longer be necessary. Until this is done, authorities establishing hospitals outside their own districts might fairly be held responsible for all cases which occur within a certain area around, and for all other cases which can be directly traced to infection from the hospital. The only exceptions should be outbreaks which could be definitely traced to some other cause than the presence of the hospital. The aggregation of a large number of cases on a limited area is also, in my opinion, a danger to the patients themselves. They are surrounded by an intensely infected atmosphere, which must retard their recovery and tend to produce fatal results.

My idea of isolation on a large scale is the provision of permanent administrative buildings, and the scattering of the patients over a large area in properly constructed and equipped marquees. The erection of these during an epidemic would involve the employment of the minimum number of men and the expenditure of the minimum amount of money. An area like that of Canvey Island would serve for London, and probably the spreading out of the hospital over a large area, and the prevention of any concentration of the infection by using tents, none containing more than, say 40 patients, would more or less completely prevent the infection being air-borne, and so obviate the disastrous effects of the present arrangements. Schemes for cremating the infective material in the air from hospitals are, in my opinion, impossible of successful excuetion. Certainly none of the plans recently suggested to be my enthusiastic inventors and others are at all practicable.



161 cases in the Orsett district during the year.

Where cases occurred in West Thurrock, they are given in these Tables in *brackets, opposite* the week in which they were notified.

A DOUBTFUL CASE OF HÆMORRHAGIC SMALL-POX.

By MR. SHIRLEY MURPHY and DR. KLEIN, F.R.S.

(*Read: May 30th*, 1902.)

The epidemiological and clinical aspects. By Mr. Shirley Murphy.

On the 9th December last (1901), I was informed by Sir Hugh Beevor that a nurse residing at a Nurses' Home, near the Strand, had died the previous night after some four days' illness. She had been seen by Dr. Frederick Spicer, who, having regard to the history and circumstances of the illness, was of opinion that the case required further investigation.

The possibility that she had died of plague could not for the moment be absolutely negatived; and inasmuch as it was matter of first importance that the body of a person thus dying should not be buried without this question being determined, I decided to make use of the opportunity the London County Council has provided for the examination of such cases, and to ask Dr. Klein to investigate it bacteriologically.

The history, therefore, of this case divides itself under two headings: the first, the circumstances of the patient and the symptoms of the disease from which she died; the second, the results of Dr. Klein's bacteriological inquiry. My own share of this paper is limited to the first, and to present the facts which were brought to my knowledge by others, for I did not see the patient during life or after death. I am, however (thanks to Dr. Spicer), able to include an account of the symptoms he observed. My colleague, Dr. Young, inquired into the circumstances of the case, and both he and Dr. Hamer took part in the post-mortem examination, and both have supplied me with the notes they have made on the subject.

The patient was a nurse (Nurse T.), aged forty-two years. For some three or four weeks she had been engaged in nursing a man suffering from a third relapse of acute rheumatism, in a house near the Strand. This man was under the care of Dr. Sing, of Southampton Street. The house was used for the purposes of a restaurant. The basement con-

sisted of cellarage, and was used for the washing and cutting up of vegetables. The ground and first floors were used as restaurant rooms; the second floor as kitchen, larder, and scullery. Above were rooms occupied by the proprietor, his wife, and servants. The house was believed to be in an insanitary condition, and Dr. Sing stated rats were frequently seen in it. A year before there had been practically a plague of rats, owing to adjacent buildings being pulled down. Rats were met on the stairs; they carried bones from one part of the house to another. Dr. Young ascertained from Dr. Sing that there had been no case of illness in this house for some time previously, except a case of ulcerated varicose veins some months before.

Nurse T. did not sleep in the house occupied by her patient. She slept in the Home, took her meals in the patient's house, but not in the restaurant: another nurse from the Home being employed on night duty on the same case, returning in the same way to the Home to sleep. Nurse T. had been engaged in this way for about two months, the last three weeks on night duty. The only known exception to this occupation of her time was a visit she made to Gravesend, on November 13th, to see a relative who had enteric fever. She slept at Gravesend one night. She may, however, have gone out in the daytime, if sent on a commission by her patient's family.

There had been no illness among the nurses at the Home during 1901, and none of them had recently been in attendance on a case of infectious disease. Dr. Young, however, ascertained that with the object of gaining experience in the nursing of small-pox, two nurses from the Home had been for a period at the small-pox hospital ships. The first of these, Nurse C., went to the ships on Sept. 2nd, left the ships on November 23rd, and went to the shelter on shore, where she remained till November 27th, and then returned to the Home. She slept at the Home one night, and then went to Halifax. She remained in good health. She was a friend of Nurse T., and they probably met at the Home.

The other (Nurse L.) went to the ships on Sept. 23rd. She left the ships on November 23rd, and returned to the Home, where she remained until November 27th, when she returned to the ships.

Nurse T. may, therefore, have come in contact with Nurse C. on November 27th and November 28th, and with Nurse L. on the four days, November 23rd to November

27th. Nurse T. began to be ill on December 4th. If infected by C., the incubation period would not be more than seven days, if by L., not more than eleven days.

Nurse T., as I have said, was unwell on December 4th. She came home, and the following morning, the 5th, was unable to get up. She felt sick and had headache. On the 6th she was no better, and thought she had influenza. On the 7th she was considerably worse, and in the absence of Sir Hugh Beevor, Dr. Spicer saw her at 5 P.M., and he writes to me as follows:—

"I found her looking really bad, with all the symptoms of high fever: temperature up, dry skin, foul tongue, sordes, fœtid breath, bowels confined, little urine, pulse rapid and feeble, respiration feeble, etc., etc., and, *most marked of all*, a peculiar deep, dusky, flushed face, with conjunctivæ bile-stained, and here and there a small hæmorrhagic spot; pupils contracted but reacted to light. This duskiness and puffiness extended up all over the scalp, and down the neck behind, and to the clavicles in front. She complained of aches and pains all over, deafness (which I attributed to the *sod. sal.* she was taking), and intense headache.

"On the upper part of the chest there was a faint rash, very faint, but still distinct, which disappeared on pressure, and which would have done for early scarlet fever, or early measles, but there were no petechial spots, no hæmorrhage, and this was late on the fourth day of the disease. Ear, nose and throat were normal . . . . . Wishing to see the rash by daylight, I went early the next morning. Sunday (December 8th), and found her much worse, evidently going to die: semi-comatose, face deeply dusky, conjunctivæ deep yellow with hæmorrhages, tongue swollen and thickly furred; trunk with chest and abdomen appeared to have got much darker, a deep brown colour, and smothered all over with hæmorrhagic spots, which gradually extended to the limbs—a typhus rash—and she died comatosed that night. The temperature, which had risen up to 104 deg. Fahrenheit, fell to 98 deg. five hours before her death."

The post-mortem was made on the afternoon of December 9th, at 4 p.m., by Dr. Hamer and Dr. Young, Dr. Klein and Dr. Allan, Medical Officer of Health of Westminster, being present. Dr. Klein took material for examination. Dr. Hamer has supplied me with the following notes, which I append to Dr. Spicer's account of the symptoms.

"On exposing the surface of the abdomen, the lower

part was found to be covered with a purpuric eruption, consisting of thickly-set pin-point petechiæ with larger blotches, some of which were the size of a split pea. The eruption was especially marked, roughly speaking, in a triangular area included between a transverse line drawn through the umbilicus, and bounded below by two lines drawn across the front of the thighs parallel to and a few inches below Poupart's ligaments. Two chains of more scattered petechiæ extended from the main area of eruption upwards towards the arm-pits. On the arms and lower legs there were purpuric blotches here and there, but the main development of the eruption was in the situation already defined. There were conjunctival hæmorrhages and minute petechiæ on the pericardium. The pericardial sac contained several ounces of fluid. The appearances of the eruption and of the conjunctivæ were those characteristic of hæmorrhagic small-pox."

In presenting this history of the case to the Society, I may add that it is in every way consistent with numerous cases of hæmorrhagic small-pox which I saw in the small-pox epidemic of 1871. There was no definite exposure to small-pox infection, but there was possible exposure to infection through the visit of one of the other nurses to the hospital ships. At the time, moreover, there were numerous cases of small-pox in the neighbourhood of the Strand and of Drury Lane. The period intervening between the beginning of the illness of Nurse T, and the return from the ships of Nurse L, was not too short for the incubation period of small-pox.

I have no information concerning the condition as to vaccination of Nurse T. When question arose as to whether her illness was small-pox, steps were taken to secure the re-vaccination of those with whom she had been associated, and there were no subsequent cases of small-pox among these people. Efforts were made, unsuccessfully, to obtain a rat from the house in Fleet Street for the purpose of examination.

The Bacteriological Aspects. By E. Klein, F.R.S.

From the body of Nurse T., at the post-mortem, twelve hours after death, blood was withdrawn from the interior of the right heart under all aseptic precautions, and this blood was used for making stained film specimens, for (*a*) plate cultivations and (*b*) for injection into guinea-pigs. At the same time, pieces of the skin from the hæmorrhagic area were taken away in preserving fluids.

Fig. 3.
Film specimen of plate culture of heart's blood of Nurse T., showing two kinds of microbes : diplococcus (streptococcus pneumoniæ) and b. myxoides. x 1000.

Fig. 7.
Film specimen of pure culture of b. myxoides. x 1000.

(*a*) The film specimens showed a large number of diplococci; in some fields of the microscope they formed chains; the diplococci were distinctly capsulated. Here and there a bacillus was seen, not capsulated.

(*b*) The plate-cultivation showed after incubation two kinds of colonies: (1) colonies uncountable in number, which corresponded in all respects to those of the diplococcus pneumoniæ; (2) a few colonies of a new and

Fig. 8.

Stab culture in gelatine of b. myxoides, after a week's growth, at 20° Cent. Natural size.

undescribed microbe, which in morphological respects comported itself exactly like the bacillus pestis, but which in culture was utterly different from it. In cultural respects it corresponded, to a limited extent, to the bacillus of Friedländer; but it differed from the latter in some respects.

This microbe, which I propose naming "bacillus myxoides," on account of its forming a slimy growth, is

pathogenic to guinea pigs when introduced intra-peritoneally, but is not pathogenic subcutaneously for this animal; it is pathogenic for the mouse subcutaneously. The first-named microbe (diplococcus pneumoniæ), which was present in the blood of Nurse T. in great abundance, proved itself indistinguishable from the diplococcus pneumoniæ also in this: that it was non-virulent to guinea-pigs, highly virulent to mice.

The accompanying lantern demonstration explains fully the nature of the two microbes.

Fig. 9.
From a section through the hæmorrhagic skin of Nurse T., showing extravasated blood in the lymph spaces of the corium; the epidermis being unchanged. x 180.

From these facts it appears, then, that this patient had in her circulation two kinds of virulent microbes, one of them being present in very large numbers: the diplococcus pneumoniæ. Now it is known from other instances in which hæmorrhages have been observed in different organs that these hæmorrhages were due to a general blood infection with the diplococcus pneumoniæ; and unless other evidence as to this Nurse having been affected with variola hæmorrhagica be forthcoming, the general infection of the blood of the circulation of this patient is quite sufficient,

in my opinion, to cause and to account for the hæmorrhages in the skin, pericardium and liver, observed in this case.

A further interesting point confirming this view was found in the microscopic sections made through the hæmorrhages of the skin of this case: the sections showed effusion of blood into the lymph channels of the skin and deeper parts of the corium; and amongst the effused blood the capsulated diplococcus pneumoniæ could be recognised in several places. Besides this, there was nowhere any indication of the formation of loculi, or any pathological change in the epidermis; the patient having been ill for four days, one might have expected some change in the epidermis if the disease had been variola.

On the one hand, then, from a bacterioscopic analysis, the general infection with the diplococcus pneumoniæ is quite sufficient to account for the disease and its lethal course; on the other hand, the microscopic examination of the hæmorrhagic spots of the skin failed to discover any indication of the variolous condition of the epidermis.

This case is, therefore, interesting to the bacteriologist and pathologist from two points: first, it yielded a microbe which in morphological, and, to a certain extent, experimental, respects might have been mistaken for bacillus pestis; and, secondly, it showed that the diagnosis of "hæmorrhagic small-pox" in this case was arrived at by indirect epidemiological evidence, and by the peculiar distribution of the hæmorrhages in the skin noticed at the post-mortem.

AN OUTBREAK OF SYPHILIS IN AN INDIGENOUS TRIBE IN INDIA.

By LEONARD ROGERS, M.D., M.R.C.P., I.M.S., Officiating Professor of Pathology, Medical College, Calcutta.

(*Read: May 30th*, 1902.)

NORMAN CHEVERS, in his *Commentary on Indian Diseases*, remarks that an outbreak of syphilis in a recently-affected tribe in India would be a very interesting study. Such an opportunity fell to my lot early this year (1901), the following account of which is based on shorthand notes of a large number of cases, made on the spot. The affected tribe was the Khonds, who inhabit the remote south-eastern corner of the tributary states of Orissa, between the north-east district of the Madras Presidency and the east of the Central Provinces: occupying a very sparsely-populated tract of dense jungle, which supplies all their primitive wants, and having very little dealing with surrounding districts, almost their only trade product being the roots of the turmeric plants. The particular group of villages which were affected was situated in a clearing of the forest, reached by a footpath, and their only dealings were with the town of Sonpur, some thirty miles distant, from whence the disease appears to have been introduced, some twenty or more years ago. Attention was recently attracted to the disease, owing to the late famine having necessitated the collection of a number of the people on a small relief work, when many of them were found to be suffering from ulcerations, and a native Civil Hospital assistant reported that the people were suffering to a large extent from syphilis, disseminated by flies and gnats; while the subdivisional officer stated that the disease had spread so rapidly among the children in an elementary school, that it had to be closed. This was all the information available before my visit.

On the afternoon of my arrival on the scene, after a journey of 120 miles from the railway, a few cases were collected from the nearest village, among which was a man with rupial sores all over the body; another with an extensive ulcer below one knee, and large nodes on the tibia

of the other leg; and a woman with ulceration and irregular thickening of the frontal bone; while in none of the rest were the lesions other than those commonly met with in syphilis. The alleged direct spread of the disease in the school remained to be cleared up; and on the following morning the children were collected, and it was found that only seven out of twenty-eight children had been attacked: two had died, two had recovered, and three were still ill. The latter presented the usual typical signs of congenital syphilis, while the two who had recovered showed evident marks of a recent attack of the same disease, their parents having also had syphilis. No direct infection could be traced among them, nor was there any evidence incriminating the much-abused mosquito. It remained to ascertain the extent of the prevalence of the disease, and any points of interest presented by it. In the course of three days, some fifty cases of the active disease were collected from the few small villages, of from three to thirty houses in this tract, in some of which as many as ten per cent. of the inhabitants still showed lesions of syphilis, although large numbers, amounting in one village to one-tenth of the population, had died during the previous year of the disease, doubtless aggravated greatly by mal-nutrition resulting from the famine conditions. A still larger number showed scars of previous outbreaks; while, as none of these poor people had used any treatment other than sulphate of copper solutions externally, and jungle roots internally, very many of them must retain the active virus in their systems; and it is no cause for wonder that the recent scarcity had caused numbers of them to break out in every loathsome manifestation of this protean affection, from numberless rupial sores to extensive ulcerations with necrosis of bones. Altogether, I doubt if much less than half the adult population were constitutionally affected.

Still worse was the state of the children: for, in addition to the large proportion with active lesions of the disease, on examining all the "healthy children" who could be collected, I found no less than 9 out of 17, or over one-half, showed evident marks of having been affected by the congenital form of the disease. Adding to these 13 cases of active congenital disease, the main symptoms were met with in the following proportions:—Out of the 22 cases, condylomata, or scars of them, were found in 21; ulcers at the angle of the mouth, or their scars, in 20; snuffles, or depressed bridge of the nose, in 19. In no case was interstitial keratitis seen, or a history of it obtained, although it

was carefully looked for, and several of the children were old enough to have suffered from this symptom. Its absence may, perhaps, be due to the much lighter strain on the eyes of these children of the forest than on their brothers under the Board School *régime* in England. In one boy the permanent upper central incisors were stunted and slightly notched, and in another the upper right central incisor only was peglike and knotched; but these were the only cases with typically-affected permanent teeth, as described by Hutchinson. On the other hand, very marked deformity of the incisor first teeth were noted in five of the young children with congenital syphilis, although not seen in any of the healthy ones. It consisted in the affected teeth being both very stunted and also much narrower than normal, so as to leave a considerable space between both of them, while they were discoloured and roughened on the surface, but none of them showed any notch. In one child, aged five, the four upper incisors and the two central lower ones were affected; in two others, aged three and six, the four upper incisors only were deformed, while in one child of three the two lower central incisors only were involved. That five out of the twenty-two diseased children showed this deformity of the teeth strongly indicates that it was of syphilitic origin, especially as none of the unaffected children had any malformed incisor teeth.

Among the adults, one of the most notable features of the outbreak was the frequency of ulcers on the soles of the feet. This may have been due to their not wearing any covering to them, and so being very liable to slight injuries when traversing the rough forest tracts. These ulcers were often multiple, and assumed the form of deep cracks in the thick horny layer which is normal in these people; and they were often situated at the edge of the sole, and caused great difficulty in walking, and much distress. The men suffered most from this affection. Very extensive ulceration below the knees, with loss of bone, were also not uncommon, some of them having lasted several years. Among curious cases was a woman with an ulcer on the left nipple, somewhat resembling a primary sore, but clearly of a late secondary nature, as she had condylomata, rupial sores on other parts of the body, while her husband and her child of three years were also affected. Another boy, of about eight years of age, showed numerous condylomatous sores, two of which formed warty growths on the skin of his penis.

Some idea of the extent to which the disease had spread may be conveyed by the following record of a group of related families. A man, aged forty, presented a secondary rash and ulcerated throat, and a scar at the base of the glans penis. Yet he denied ever having had a primary sore: as indeed did all these forest men, differing in this respect from townspeople. His wife and child are said to be healthy, his infection being evidently recent. In the same village live three nephews of this man. The first is aged about thirty, and now has ulcers on the soles of his feet and round the knees. His wife has condylomata in the armpits, numerous rupial sores, and an ulcer on the left nipple. She has had two children, one of whom died with symptoms of congenital syphilis in childhood, and the other, aged three years, now shows ulcers at the angles of the mouth, snuffles, narrow, stunted, peg-like lower two central incisor teeth, and enlargement of the liver and spleen, with fever—although the latter may be malaria, which is very rife at this place. With this family another nephew, aged seven years, lives, and he has been ill two years, commencing with a rash all over the body. He now shows condylomata and rupial sores, and snuffles. The third nephew lives in a separate house. He is aged about 30, and has been ill about three years, and now shows numerous rupial sores. His wife shows ulcers on her feet, and numerous scars of old ulcers on different parts of the body. She has had two children, one of whom died when one year old, and the other when three, both with symptoms of congenital syphilis. In the same village are seven other households, not related closely to the above families, and they are all said to be free from the disease. The above-described group affords a terrible picture of the ravages of untreated syphilis among an ignorant people.

The details already given are sufficient to indicate the terrible extent of the disease among these people; but it is also of interest to determine how long syphilis had been present among them, and whence it came. The histories of different cases clearly showed that the disease had been present somewhere about twenty years. One man gave a history of rash and sore throat, followed by rupial sores, beginning twenty-five years ago; and he now had a very large ulcer just below the knee, of three years' duration. The calculations of these people, however, cannot be implicitly relied on. Of more importance is the evidence derived from the following two interesting cases of late

manifestations of the congenital disease in a brother and sister, aged twelve and eighteen years respectively. Both their father and mother suffered from syphilis before they were born, according to the mother herself, who presents numerous scars of old ulcers round the knees, on the feet, and other parts. Five other children died young, particulars of which could not be obtained. The daughter suffered from ulcers at the corners of the mouth, and snuffles when an infant, and broke out in ulcers leaving scars when five years old. Two years ago, large ulcers broke out round the right knee, which lasted one year, and the resulting scars have caused some deformity of her right foot by their contraction. The boy, now aged twelve years, suffered from rash, ulcers round the mouth, and snuffles when a child. Last year, several large holes appeared on the bones of both legs, and a small one on the right ulna, which still persist, and present a very characteristic appearance. Neither of them show any affection of the teeth. These cases were clearly of congenital origin, and their parents must have suffered from syphilis about twenty years ago. It is not quite so certain whether the disease may not have been present much more than twenty years, but several of the old men were positive that it was unknown within their memory, and that it had been introduced from Sonpur somewhere about twenty years back, when trade with that place began to bring them into contact with the Sonpur people; and this statement is borne out by the fact that syphilis has been very prevalent in the town of Sonpur for many years past. The evidence, then, points to the disease having been introduced to this group of villages about two decades ago, during which time the majority of the people have become infected by either the constitutional or congenital disease, partly owing to their low standard of morality, and direct infection from crowding and uncleanliness; and no specific treatment ever having been adopted, the recent famine conditions so lowered their vitality as to allow the virus to manifest itself actively in a number of them, with a resulting high mortality and the production of the terrible series of cases which I saw. A dispensary is now being started for their treatment; while, fortunately, the infected community is a small and very isolated one.

FILARIASIS IN THE WEST INDIES AND ITS PREVENTION.

By GEORGE C. LOW, M.A., M.B., C.M.(Edin.); Craggs Scholar; London School of Tropical Medicine.

The life-history of *Filaria nocturna*, the nematode worm that is responsible for the large group of diseases classified under the term "Filariasis," has now been completely worked out, and the gradual steps that have led up to this knowledge have now passed into the realm of ancient history: a history, however, which is so interesting that we may briefly refer to it here in the preliminary part of this lecture.

Bibliography.

As long ago now as 1863, Demarquay, in Paris, first discovered the embryo of *Filaria nocturna*. One day, on microscopically examining some fluid removed by paracentesis from a lymphatic swelling of the scrotum in a young man from Havana, he was astonished to find several very minute cylindrical worms amidst the cellular structure and other *débris*, and he published this remarkable fact in the *Medical Gazette* of Paris* of the same year, giving a very fair illustration of the parasites as well.

Strangely enough, his discovery attracted no attention, and in the course of a few years it appears to have been totally forgotten and lost sight of.

Wucherer, in Bahia, in ignorance of this paper, rediscovered the embryos, however, in cases of chyluria in 1866; and after collecting a series of twenty-seven such cases, in all of which he found similar parasites, published the results in the *Gazzeta Medica de Bahia* in 1865 and 1869.†

No further advance of any great importance was made in the subject till Lewis,‡ in India, in 1872, found similar

* *Gazzete Médicale de Paris*, 1863, chap. xviii, pp. 9, 665.

† *Gazzeta Medica de Bahia*, Dec. 5th, 1868, and Sept. 30th, 1869; *und Lenckart's Parasiten*, Band II, pp. 9, 646.

‡ Eighth, Tenth, and Fourteenth Annual Reports of San. Com. with Government of India.

embryos in the blood of a patient suffering from diarrhœa and emaciation; these he named, on account of their peculiar situation, the *Filaria sanguinis hominis*, a nomenclature which was adopted and retained till quite recently, when Manson, on discovering that this was only one of many worms that inhabit the blood of man, changed it to *Filaria nocturna*.

After Lewis's investigations everyone working at the subject agreed that these small, actively-moving worms seen in the blood must be the young or embryonic forms of adults, lying somewhere or other in the body of the host, and observers began systematically to search for them.

The honour of discovery eventually fell to Bancroft, in Brisbane, Queensland,* as, while opening a lymphatic abscess in the arm of a patient one day, in whose blood filarial embryos had been demonstrated, he found a small worm, 3 inches to 4 inches long, and about the thickness of a human hair. Soon afterwards he obtained more from another case, sending them to Cobbold, in England, for identification;† and the latter authority, on examining them, found embryos in the uterus of one in shape and measurement exactly similar to specimens seen in the blood of other cases, and so came to the conclusion that the young forms found swimming free in the blood of people suffering from filarial disease were the offspring of parent worms living in the lymphatic trunks of man.

In 1878 your President, Dr. Manson,‡ in Amoy, China, made the very remarkable and far-reaching discovery that certain species of mosquitos act as the intermediate hosts for the further development or metamorphosis of the embryonic *filariae*. Being struck by the fact that the embryos in the blood showed no evidence of growing or in any way changing, he was led to the belief that in some way or other they must enter an intermediate host to allow of their further development: a mode of procedure so common amongst the various forms of parasitic worms.

Starting on this assumption, he began to examine different suctorial insects which were known to have fed on infected cases, and amongst these mosquitos formed the large majority, as they were very prevalent in the district. In all of those examined he found that the *filariae* were

* *Lancet*, July 14th, 1877, and Cobbold, *Parasites*, 1879.

† *Lancet*, October, 7th, 1877.

‡ *Lancet*, May 12th, 1878; *Proc. Linn. Soc.*, March 7th, 1878; *Medical Times and Gazette*, London, 1878; *Transactions*, Linn. Soc. of London, 2nd Series; *Zoology*, vol. ii, Part x, p. 367.

ingested with the blood into the stomach, but that in most of them they were slowly digested and disappeared; the exception to this rule being the case of a small brown unstriped mosquito, nocturnal in its habits.

In the case of this insect the embryos, instead of dying and gradually disappearing, bored their way into the tissues of the host, chiefly the thoracic muscles, and there underwent a most remarkable development. To follow these changes out accurately, a Chinaman, who had filarial embryos in his blood, was persuaded to sleep in a specially-erected wooden framework, covered with mosquito netting, the door of which was left open for a short time each night to allow of mosquitos entering to bite; and then in the mornings the gorged insects seen hanging on to the netting were carefully caught and put in stoppered bottles, being kept there till required for dissection. Three species of mosquito were in the habit of visiting the house: two of the variety known as the "tiger," sooty in colour, with white bands on their legs, while the third was a small brown mosquito called the *Culex pipiens*, but which our present knowledge of tropical mosquitos would now clearly indicate to be *Culex fatigans*, the common domestic or house-mosquito of the tropics. The former two proved to be inefficient, but in the latter the embryos migrated to the thoracic muscles and there underwent a metamorphosis, attaining in about eight days an enormous size, $\frac{1}{16}$th of an inch in length by $\frac{1}{800}$th of an inch in breadth, and, further, now possessing a well-developed alimentary canal and other structures. As most of the insects died, about this time, Dr. Manson put forward the view that in Nature the mosquito, after laying its eggs on water, died there and disintegrated, thus freeing the young embryos, which could presumably live in this medium until taken up by man, when they would by passing through the stomach gain some lymphatic trunk, and there become the mature parasite again.

Confirmation of this wonderful metamorphosis soon appeared. Lewis,* in India, on beginning a systematic research into the subject, was astonished to find that, when care was taken, 14 per cent. of the mosquitos taken at random around his house contained filarial embryos. He followed out the different changes undergone in the muscles of the insects, but did not describe forms later than the fourth or fifth day, most of his mosquitos then dying.

* *Proc. Asiatic Soc.*, Bengal, March, 1877, pp. 9, 89; and Fourteenth Annual Report of the San. Com. with Government of India, 1878.

Insino,* in Egypt, in 1882, also did similar work, but failed to find such advanced forms as the former two observers.

For many years matters remained in *statu quo*, till Grassi,† in Italy, in 1890, indirectly confirmed the insect phase of the filaria of man, by demonstrating that the embryos of *Filaria recondita*, a blood-worm of dogs, undergoes a similar metamorphosis in fleas. Briefly stated, the parental forms live in the fatty tissues round the hilum of the dog's kidneys, their embryos passing from there into the blood; and then, when fleas, especially *Pulex serraticeps*, feed on such infected animals, they ingest the parasites into their stomachs.

From there, the embryos pass through the stomach, embed themselves in the fat-cells, and undergo four stages of development, eventually becoming almost mature. Infection experiments by feeding dogs with such fleas gave negative results.

In 1899, Bancroft (junr.),‡ in Australia, worked out afresh the metamorphosis of *Filaria nocturna* in the mosquito, and verified completely the previous discoveries, at the same time amplifying them, and suggesting other important details.

Before commencing his work, he made a special study of the habits of mosquitos, and found that to keep such insects alive for any length of time, it was necessary to feed them on suitable juices or fruits.

He began his series of experiments by first rearing large numbers of young mosquitos from eggs taken from water; and, when those became insects, they were fed on a girl with *filariae* in her blood. Serial dissections of such insects, day by day, showed that the final metamorphosis in Australia was reached by the sixteenth or seventeenth day; most of the mosquitos easily living for this period of time when fed on bananas and similar fruits.

Of more importance, however, than these results, were his experiments on dissecting mosquitos with metamorphosed embryos in them in water, so as to allow the contained parasites to escape into that medium. He found that they only lived for three to four hours, and so came to the conclusion that water was injurious to them, and not the medium by which they are transmitted to man, the

* *Medical Times and Gazette*, May 27th, 1882, p. 554.

† *Centralbl. für Bakter. und Parasiten*, Band II, No. I, 1890; also John Hopkins, *Hospital Reports*, vol. viii, 1892 (Nuttall).

‡ *Journal of Tropical Medicine*, London, November 15th, 1899, p. 91.

final host. On those grounds, he brought forward two theories: firstly, the accidental swallowing of mosquitos, which seemed highly improbable; and, secondly, a suggestion that the *filariae* might come out by piercing the œsophagus of the mosquito when it was sucking blood, and so get into the system of man through the skin. About the same time, Bancroft made a large collection of filiariated mosquitos, preserved them in glycerine, and sent them home to Dr. Manson in London.

While working in the London School of Tropical Medicine, on this material, in the spring of 1900, I was able to demonstrate the path* by which the young *filariae* escape from their intermediate host, the mosquito; namely, through the proboscis of the insect, thus disproving the water-transmission theory, and making filariasis a directly inoculable disease. These conclusions were reached by making serial sections of mosquitos with a microtome after embedding the insects in celloidin. By this means, it was found that the young *filariae* after reaching their highest stage of development in the insect, instead of lying passively in the thoracic muscles, leave that tissue, and travel forward in the direction of the head of the mosquito, passing into the loose cellular tissue which abounds in the prothorax, in the neighbourhood of the salivary glands. After a short stay there they pass into the head, remain for some time below the cephalic ganglion, and then pass into the proboscis, where they lie awaiting an opportunity of gaining access again to man, when the mosquito re-bites.

Working, simultaneously, in India, Captain James,† of the I. M. S., while dissecting fresh mosquitos, also found a completely metamorphosed *filaria* in the proboscis of one of those insects.

The proboscis view was, later in the same year, corroborated by Grassi,‡ in Italy, who, along with Noe, traced the embryo of *F. immitis*, one of the blood-worms of the dog, through the common *Anopheles maculipennis*. He found that the embryos developed in the Malpighian tubes and not in the thoracic muscles, and in about ten days became completely metamorphosed, migrating to the head of the insect, and so into the proboscis. By direct infective experiments on healthy dogs, he found that the worms passed from the proboscis into the tissues of the dog, and dis-

* *British Medical Journal*, June 16th, 1900.

† *Ibid.*, September 1st, 1900, p. 533.

‡ *Ibid.*, November 3rd, 1900.

appeared. His views on the exact position of the parasites in the proboscis, and how they escape, differ slightly from mine; but they in no way interfere with the main issue, namely, the fact that the worms pass directly into the human host.

General Geographical Distribution.

The geographical distribution of *Filaria nocturna* is tropical, and, to a lesser degree, sub-tropical. Its greatest prevalence is between the latitudes of 30 deg. north and 30 deg. south; that is, roughly speaking, in those regions with a higher mean temperature than 70 deg. Fahr., though it is also found in the sub-tropics, where the temperature falls short of this mean. The probability is, however, that in such places as Charleston in North America, Carthagena in Spain, Brisbane in Australia, it has been introduced by the sea from the tropics, and, finding a sufficiently high temperature at part of the year for its development in the mosquito, has remained and become indigenous. It has been found and reported on in many of the tropical regions in Asia, Africa, North and South America, Australia, and also in many of the islands. In the New World, it has a wide range, from Charleston in the north to the Brazils in the south: this including the West India Islands, from Cuba to Trinidad, the part of the world which specially concerns us.

The Life-History of Filaria Nocturna.

Intracorporeal Phase.—The life-history of the parental forms of *F. nocturna*, and of its embryos which inhabit the blood-stream, is so fully described now in the text-books on tropical pathology,* that we need only glance at it here in passing.

The parent *filariae*, long hair-like transparent nematodes, 3 in. to 4 in. in length, live in some part or other of the lymphatic system of man: sometimes in the thoracic duct, sometimes the pelvic lymphatics; or, again, in the distal lymphatics of the limbs. The female is larger than the male, measuring 3 in. to 4 in. in length; *i.e.*, 76 mm. to 100 mm. by $\frac{1}{90}$th to $\frac{1}{125}$th of an inch, .288 mm. to .211 mm. in breadth, and they are visible to the naked eye, resembling fine catgut or a piece of white thread. In both cases

* Manson, *Trop. Diseases*, second edition, 1900.

the head is club-shaped, with a simple circular mouth, and is joined to the body by a somewhat narrow neck. The body-cavity, enclosed by a longitudinal layer of muscular fibres, contains a well-developed alimentary canal, and the generative organs, which are well developed in both sexes. The uterus of the female becomes filled with ovoid-shaped eggs, out of which a small embryo appears; and these, after being born alive into the lymph-stream, find their way *viâ* the thoracic duct into the general blood-circulation. It has not yet been determined how long the adults are capable of living, but it is to their presence that the various pathological lesions found in filariasis are attributed. Their death and decomposition often act as the nidus for abscess-formation; and, by augmenting inflammatory changes in the lymphatic vessels, cause the well-known filarial lymphangitis, or even the more terrible disease, elephantiasis.

The embryonic forms in the blood may easily be demonstrated by taking a drop of blood from an infected person at night, and examining it under the low power of the microscope. It will then be seen that they are minute little creatures, like miniature snakes or eels, and can be watched wriggling about amongst the red blood corpuscles. With a high power they appear cylindrical, with a rounded head and a tapering tail; have a mouth and a central column of cells, are enclosed in a fine sheath; but, apart from those features, very little more can be made out. By day they disappear from the peripheral blood collecting in the blood-vessels of the lungs; but as night comes on they reappear, thus forming what Manson has termed "filarial periodicity."

Extracorporeal Phase.—Naturally, every blood-sucking insect that imbibes the blood of a filarial patient at night must necessarily swallow large numbers of embryos; but the large majority of such insects do not act as efficient hosts; *i.e.*, they are incapable of allowing of the development of the *filariae* in their tissues; as the blood is digested so are the parasites, and they entirely disappear. A second class are what may be termed partially efficient: in their case the embryos not only migrate from the blood contained in the stomach to the thoracic muscles, but even undergo a slight development, which is, however, slow, and ends in nothing, death and absorption eventually taking place. Some species of mosquitos act in such a manner. The next, or third, class, are the completely efficient host, as in them the complete metamorphosis of the *filariae* takes place, and

eventually they are capable of re-infecting man. Several genera of mosquitos, as far as our knowledge at present goes, come under this category.

Manson* found a small brown mosquito in Amoy, called the *Culex pipiens*, but which probably was *Culex fatigans* efficient; while Bancroft, junr.,* in Australia, found *C. ciliaris*, the common house-mosquito of those parts, to be the same. James, in Travancore,* found *Anopheles Rossii;* the second expedition of the Liverpool School of Tropical Medicine to the West Coast of Africa found *Anopheles costalis;*† Daniels,‡ in Central Africa, found *Panoplites Africanus* (Theobald); and working in the West Indies I have found *Culex fatigans*§ and *Anopheles albipes* (Theobald). It is quite possible as time goes on more may be incriminated, but even without such being discovered it is seen that quite a large number can act as efficient hosts for disseminating the disease.

Though almost completely mature forms can be detected in the thoracic muscles of *Anopheles albipes*, a common West Indian mosquito, nevertheless the chief carrier of the infection, as far as the West Indian islands are concerned, is *Culex fatigans*, which along with *Stegomyia fasciata*, forms the common domestic or house-mosquito of those parts. This being the case, we may trace the metamorphosis of the embryo *filaria* through *Culex fatigans*; as, though the time noted by me as taken in St. Lucia varies somewhat from that described by other observers in different parts of the world, yet the changes undergone may be taken as typical of the whole class.*

Starting with mosquitos reared from larvæ, so as to make sure of an uninfected stock, the mature insects were allowed to feed at night on a negro with numerous filarial embryos in his blood, and were then kept in houses, with bananas for food, till required for dissection.

Twelve to twenty hours after feeding, large numbers were seen to have arrived in the muscles, and even in forty hours showed a considerable amount of change, there being an increase in the number of cells, and a fine cuticle tapering to a point posteriorly. By the end of four and a half days the sausage stage is reached, the alimentary canal has begun to form, and the mouth and

* *Loc. cit.*

† *British Medical Journal*, "Malarial Expedition to Nigeria," Jan., 12th, 1901, p. 97, and *British Medical Journal*, Sept. 7th, 1901, p. 612.

‡ *Jour. Trop. Medicine*, London, June 15th, 1901, p. 193.

§ *British Medical Journal*, June 1st, 1901, p. 1336.

anus are visible. From this time growth goes on with great rapidity, and at six and a half days the embryos have lengthened still further, showing now a mouth, an anus, and an alimentary canal quite distinct and well formed. On the following day the œsophageal bulb appears, most have lost the fine cuticle, and some show the commencement of the trilobed tail.

At eight and a half days they measure 0.88 mm. in length by 0.04 mm. in breadth, the large cells which originally formed the intestinal tube having at this date become transformed into five small cells, and the whole appearance very much resembles that seen in the final stage of development.

At ten and a half days, forms measuring 1.26 mm. ($\frac{1}{20}$th in.), to 1.3 mm. in length by 0.04 mm. in breadth, are seen. Anatomically they present a mouth, an œsophagus, an œsophageal bulb, and a long alimentary canal ending at the anus, which is situated some little distance above the trilobed tail. Movement is now very active; though they do not change their positions on the slide, they are constantly twisting and coiling themselves about in what has been called a purposeless manner, very much resembling the movements of the immature embryos when seen in a drop of blood.

By the following day, eleven and a half days metamorphosis is complete, and mature forms are seen in the head and neck, and proboscis, and more rarely in the abdominal cavity. The embryos vary somewhat in length, and the muscles may show a number of short *filariae* that have developed more slowly. The largest now measure $\frac{1}{16}$th of an inch in length, 1.60 mm. by .024 mm., .032 mm. in breadth; the mouth is still simple. An œsophagus, expanding into a bulb, opens into the intestine, which was seen previously to end in an anus some distance above the trilobed tail. A musculo-cutaneous layer is seen under the cuticle, and there is some differentiation of cells to form the generative organs, not sufficiently developed, however, to tell the sexes from each other.

When the mosquito next bites man, any *filariae* situated in the proboscis pass from this site under the skin of the person punctured, and then make their way to some lymphatic trunk, where they increase still further in length, become sexually differentiated, and so re-start their complicated cycle of life.

Prevalence of the Disease in the West Indies.

In estimating the prevalence of filarial disease in any given area, it must be remembered that the majority of persons affected show no clinical symptoms whatsoever; and although one may get a rough estimate by noticing the number of cases of elephantiasis and other filarial complaints in hospitals and poor-houses, the only way of accurately determining the approximate amount of infection is to examine the blood of as many of the population as possible at nights for filarial embryos. This, though a tiresome task in many instances, is easy, and the blood of a hundred individuals or more may be taken each evening between 9 P.M. and midnight, the subsequent examination being left over till next morning.

The results of the statistics collected from British Guiana and some of the West Indian islands show that, as a whole, those places have a smaller percentage of the disease than many other parts of the tropics, especially than some of the Oceanic islands in the South Pacific.

When one, however, considers the islands, and British Guiana on the mainland, separately, it is at once seen that each individual place varies very greatly, one island, even from the next closely adjoining it, the more or less flat and cultivated ones having a much larger amount of the disease than those which are hilly and mountainous.

Of all the places specially examined, St. Kitts, one of the leeward group of the lesser Antilles, easily stands first, no fewer than 32.8 per cent. of the inhabitants suffering from filariasis.

The island is of volcanic formation, consisting of a central core or ridge of mountains, with the land sloping gently from this to the sea, this being sandy and porous in nature, and extensively cultivated with sugar cane.

Even in the wet season, with the exception of some swamps or low-lying ground on the windward side of the island, very little water stands in the country districts, large watercourses carrying it quickly away to the sea.

The town of Basseterre, situated on flat sandy ground on the seashore near the end of the central ridge, is typically West Indian, native huts and dwellings being intimately mixed up with the houses of the better-class white population, and in this area *Culex fatigans*, the common domestic mosquito of those parts, is very prevalent, breeding chiefly in the privies with which the town is abundantly supplied, and also in other collections of water so common in West

Indian towns and villages, and which we will deal with later under the prevention of the disease.

Dominica and St. Lucia, the two most mountainous islands of the group, suffer comparatively slightly, only having 7.63 per cent. and 7.58 per cent. respectively, and the clinical manifestations correspond, few cases being treated annually in the hospitals, and few individuals suffering from elephantiasis being seen about the streets and other places. Their chief towns (Roseau in Dominica, and Castries in St. Lucia) are situated on the leeward coasts in hollows, shut in all round by high mountains, and are very hot with little breeze, and many native dwellings are found all over the centre and outskirts.

Stegomyia fasciata, one of the domestic mosquitos of those parts, an inefficient host for the filarial parasite,* is commoner than *Culex fatigans*, and this may account for a corresponding diminution of the disease.

Barbados, the most easterly of the Caribbean Islands, consists of a flat coral formation, only attaining to about 1000 ft. above sea-level at its highest point. For its size, 21 miles long by 11 miles broad, it is very densely populated, containing no fewer than 200,000 inhabitants, about 30,000 of whom live in Bridgetown, the chief town of the island. It is peculiar in having no proper rivers, very little standing water of any kind, and *Anopheles* mosquitos and malarial fevers are non-existent;† on the other hand, however, filarial disease is very common, 12.66 per cent. of the people suffering from it. With its dense population, chiefly negroes, and their persistent habit of storing water in barrels and other similar receptacles, which afford suitable places for *C. fatigans* breeding in, this is not surprising. Before the water supply was brought into the town there is no doubt that the disease was much more prevalent than it now is, and elephantiasis was then so common as to give rise to the local name of "Barbados leg" for this complaint.

The next island, St. Vincent, mountainous and hilly, with a sparse population, has only 6 per cent. of its inhabitants infected; and Grenada, to the south of this, enjoys the enviable distinction of only having .56 per cent. of the disease; the clinical manifestations again bearing this out, as cases of filariasis are practically never got in the hospitals, and the medical men throughout the island have

* *Loc. cit.*

† *British Medical Journal*, Sept. 4th, 1901, and *Jour. of Trop. Med.*, London, Sept. 2nd, 1901.

very rarely seen it. Why this should be is rather difficult to appreciate, and is very interesting. The island, it is true, is mountainous, and St. George's, the chief town, is largely built on the slopes of the hill-sides overlooking the harbour; but even then there are plenty of native huts where water is often kept standing for long periods of time, and *C. fatigans*, though not perhaps numerous, still exists. Here we have, then, a tropical island, with a suitable intermediate host present, and sources of infection coming in by the immigration of Barbadians and others, and yet the disease never seems to have taken root and spread. That it can live in the island is proved by cases of elephantiasis occurring in people who have never been out of the island.

Trinidad, to the south of Grenada, close to the mainland, gives a percentage of 10.75 per cent.; that is somewhat less than Barbados. Port-of-Spain, its chief town, has a very mixed population, and has plenty of native huts in its immediate vicinity. It is a fine town, well built and well drained; but, even with this, *C. fatigans* is common, and tanks and other breeding-places are far from rare.

Leaving the islands now, and passing to British Guiana, situated on the mainland of South America, some very interesting features are to be made out in connection with the disease. *F. nocturna*, which is much more abundant (viz., 22 per cent.) than in the other islands, with the exception of St. Kitts, is only got in the towns and coast-line, and not in the primæval forests which clothe the interior. East Indian immigrants also, who live chiefly in the country on the sugar estates, enjoy a much greater immunity than the Negroes, Creoles, Portuguese, and others who live in the towns, where the percentage given above was calculated.

Georgetown, the chief centre of the colony, of course affords splendid breeding-grounds for all sorts of mosquitos, trenches or canals existing in many of the main streets; and further, as the water-supply is bad, the lower classes have to store rain-water in barrels and other situations for their drinking supply. This being the case, the breeding-grounds for the intermediate host, *C. fatigans*, are simply legion; and those operating on a dense population, quickly spread the disease: the wonder being that more does not exist with circumstances so favourable for its spread.

All types of the disease are met with in the West Indies, the most infected places showing more of the rarer manifestations.

Filarial lymphangitis, locally termed in Barbados "fever and ague," the "elephantoid fever" of Fayrer, is very common in Barbados and St. Kitts, and very few of the white people, who have been born or lived most of their lives in those islands, have escaped attacks at one period or other of their existence. Lymph scrotum, vaccine groin glands, and abscesses due to the *filariae*, are all common throughout the West Indies, as is also the case with elephantiasis. Affections of the lower limbs are the most frequent site of this unsightly complaint; but, in the intensely filarial places, Demerara and St. Kitts, it is often seen in the scrotum, penis, prepuce, breasts, ears, and also in the form described as the pedunculated groin elephantiasis by Daniels* in Demerara and Corney† in Fiji.

The Prevention of Filariasis in the West Indies.

The prevention of this disease, as far as some of the West Indian islands are concerned, is simple, not only theoretically but practically. The dangerous class of case to the community at large is very often not the one that exhibits pathological symptoms, as in almost all cases of elephantiasis, and in many cases of filarial lymphangitis, the embryos disappear from the peripheral blood; but the one that exhibits no definite symptoms at all, through having his blood infected with parasites. It is clear, therefore, then, that everyone with filarial embryos circulating in his blood may spread the disease to other people in the same house and vicinity—not directly, but by infecting the special species of mosquitos which are efficient to allow of the development taking place in them. These infected mosquitos live and frequent the house, biting fresh people every three days or so; and, eventually, when the parasites have arrived at maturity, and are situated in the proboscis, re-inoculate the original person again, or inoculate fresh individuals.

As a means of personal prophylaxis, then, every one in a tropical climate should always sleep under a properly-applied mosquito net. If he should happen to harbour embryos in his blood, then he will not infect mosquitos; and conversely if he is free, he will not be bitten, and so inoculated by infected mosquitos. It is an astounding fact, that though Dr. Manson's original discovery of the part played by mosquitos in the spread of this disease dates back to 1878, that very few of the people permanently

* *British Guiana Medical Annual*, 1896. † *Lancet*, April 6th, 1889.

residing in the West Indies ever use mosquito netting. In a long series of cases of the disease in the better-class white population in Barbados and St. Kitts, the usual answer to the question of "Do you sleep under a mosquito net?" is "Oh, no, mosquitos never bite me; and besides, they are hot and stuffy." The proof of the fact that mosquitos do bite them is, that they are infected; and the question of the heat is only a loophole for escape.

It is really a matter of carelessness and lethargy, which is so apt to appear in people permanently residing in those tropical parts; and this is aided by the fact, that though they are nightly bitten by mosquitos, very little irritation or mark is left on their skins, a gradual immunity to the effects of the bites being obtained by prolonged residence in the climate. It follows that a much more careful use of this easy means of personal prophylaxis for the wealthier classes would help to diminish the disease, and would prevent much of the useless suffering that is at present undergone by them.

This method of prevention is, of course, only palliative, and is useless for the bulk of the native population, who would never adopt it; so the real *crux* of the question turns on the destruction of the intermediate host, the mosquito, without which the disease could not go on.

Culex fatigans and *Anopheles albipes*, I have shown you, are the two disseminators of filariasis in the West Indies; and, as the latter is, firstly, non-existent in Barbados, and, secondly, is non-existent in most of the towns of the different islands—with the exception of Georgetown in Demerara—places where most of the filarial infection is found, and the disease commonest, the whole matter therefore turns on the abolition of the former. To get rid of this pest about houses is comparatively easy, if the action is universal, and if their life-history is studied to see which is the most vulnerable point to strike them at.

As the habits and breeding-grounds of *Stegomyia fasciata*, an inefficient host for *F. nocturna*, but the host for yellow fever, are identical with the one we are considering, and, as they co-exist in all of the islands, the means adopted for the destruction of the one will equally apply to the other.

Culex fatigans, the common domestic mosquito of the West Indies, then, is a small, brownish-yellow insect, the males being harmless; *i.e.*, not biting, and being distinguished by their more slender build and feathery antennae.

It is essentially a domestic mosquito, not being found far

from houses, and is nocturnal in its habits; sleeping by day in the darkest part of the room it can find, and only becoming active just as darkness begins to appear. Its presence when near one can be readily detected by its humming or piping noise; and then, choosing an exposed part, it gently settles, and proceeds to bite, remaining if not disturbed for a considerable time, sucking up the blood. After its meal, it is gorged and heavy, and rising slowly, seeks some quiet spot where it may sleep off the effects of its meal; remaining here more or less torpid for a day or two, till all the blood is digested, and then it is ready to start afresh.

The eggs of this species are deposited in boat-shaped masses, in any artificial collections of water, such as tanks, water-barrels, wells, tubs, cisterns, and gutters.

In about two days each egg gives rise to a small larva, which swims about in the water, grows rapidly, and feeds on the animalculae and other organisms found there. They breathe by a long tube at the posterior end of the body, and have to come to the surface of the water to allow of the contact of the air, their position then being perpendicular, or at an angle with the head hanging downwards.

In twelve to fifteen days, according to the temperature, they change into the pupa or chrysalis; they now cease to feed, and the respiratory tubes become altered in position, now being found in the dorsum of the thorax. Two days are spent in this phase, and then the skin splits down the back, and the imago or perfectly-formed insect emerges, rests for a little on its cast-off case, and then flies away.

To diminish or to completely eradicate mosquitos, then, from a house, by striking at their larval stage, two ways are open to us: Firstly, either to have no collections of water for them to breed in; or, secondly, if such cannot be entirely done away with, to render it harmful to the insect, so that it can no longer develop in it.

In Barbados and St. Kitts, where there is now an abundant pipe water-supply, and plenty of stand-pipes exist at suitable places for people drawing water from when they require it, no standing water, such as water-barrels, wells, tanks, or other collections should be allowed at all; and this I have strongly represented in popular lectures to the Sanitary Inspectors and general population, and officially to the Governments of those islands. The expense would be trivial, and can be easily included in the estimates of the general sanitary work; and I am happy to

state that St. Kitts, with praiseworthy energy, has started at once in the matter, and Barbados is also following suit. For the other islands, where the water-supplies are not so good and not so generally applied, and for Demerara, which is almost totally dependent on a rain-water supply for drinking purposes, a certain amount of water must be stored, but there is no reason whatsoever that this should not be stored properly, and useless accumulations abolished.

I have proposed for Barbados and St. Kitts that a law should be passed authorising the sub-inspectors of sanitation, when they see barrels or tubs of water full of mosquito larvæ on their daily rounds, to have those emptied, and to impress on the individuals responsible the danger, and to see that it does not occur again.

The whole question is really one of sanitation; larvæ of mosquitos should be looked on as insanitary products, just in the same way as fæcal excrement, garbage and offal, and removed accordingly.

In towns where rain-water has to be collected for drinking purposes, as in Georgetown, Demerara, and on the Morne, at St. Lucia, where there is a military garrison, the water should be stored in iron tanks with perfectly tight-fitting roofs, and the doors for getting in to inspect and clean the tanks should also fit tightly, no apertures large enough for mosquitos entering existing. Water-barrels for a similar purpose could easily have mosquito-proof metallic netting applied over them; but then the poor population cannot probably afford this—or, what is more likely, are too careless and uneducated to think of such a thing.

It is a common habit amongst Barbadians to keep tubs of water in their gardens for growing water-lilies; and after one of my lectures an individual asked me if he could still keep those, as he had minnows in them, and never saw any larvæ swimming about.

I personally inspected his tubs, and found that he was correct in his statements; and, further, on procuring larvæ from another source, and introducing them beside the minnows, the latter immediately ate them up with great avidity. Other experiments conducted with gold fish gave similar results, so this affords us an easy and ready means for destroying mosquitos in certain varieties of water which it would be difficult or inconvenient to have absolutely done away with. Ornamental fountains, large tanks, tubs for growing water-plants in, ought therefore to have those small minnows or gold fish introduced, and they will then be mosquito-proof.

Privies, catch-pits, and old wells abound in all West Indian towns and villages, affording breeding grounds for many mosquitos; and those, in addition, being very insanitary, should be completely filled up, the earth system being adopted for number one, drains for number two, and nothing for number three, as they or any other substitutes are unnecessary.

Kerosene oil is useful in many cases, but is only temporary, costs money, and will, therefore, not become of universal use amongst the poor. Radical methods are much better for domestic collections of water than temporary ones, as they are almost as easy to carry out, and are permanent when accomplished.

By such a simple and inexpensive procedure, a house which was before infested with mosquitos can, in a very short time, be rendered almost entirely free.

It is by striking at the larval stage that we can hope for the best results; the destruction of the adults by fumigation and other means is much more troublesome and expensive, and is at the same time practically impossible, especially if a collection of water near at hand is constantly pouring in new generations of mosquitos.

The whole question of putting an end to disease by the destruction of mosquitos is new to the West Indies, and a considerable amount of opposition is met with in some of the more ignorant and superstitious of the islands.

Such arguments, however, proceed from more or less uneducated laymen who are ignorant of the scientific work done on the subject, and are really of no value, though in some instances they act prejudicially on the intensely stupid negroes.

As I have already said, St. Kitts and Barbados have taken up the subject, and there is no doubt that the other places will follow their example.

The Colonies ought now to know their danger and the means of combating it, and each Government must apply itself to the matter, spending a certain amount of money gradually in relation to the funds at their disposal, till they eventually free themselves from this loathsome and disgusting disease: one which is responsible for largely filling almshouses and other charitable institutions, and which brings much useless suffering to many of those unfortunately affected by it.

OBITUARY.

JOHN WICHENFORD WASHBOURN, C.M.G.

BY the death of Dr. Washbourn, on June 20th, 1902, our Society sustained a most grievous loss. At the time of his death he was one of the General Secretaries, which office he had held since 1898. He contributed more than one paper to our *Transactions*, and frequently took part in our discussions. Of the many societies of which he was a fellow or member there were none in which he took a deeper interest than the Epidemiological; being a thoroughly trained and equipped bacteriologist, as well as an experienced and acute clinician with a special knowledge of infectious diseases, he was particularly attracted by all questions relating to the origin and spread of epidemic disorders.

He was born at Gloucester on June 22nd, 1863, and came of a family, the Washbourns of Wichenford and Washbourne, in the counties of Worcester and Gloucester, whose pedigrees can be traced back to the fourteenth century. He was educated at King's College, Gloucester. On leaving school in 1880, he entered Guy's Hospital, where his career as a student was exceptionally brilliant, chief among his many successes being the Exhibition in Organic Chemistry and the Scholarship in Medicine at the London University. He graduated M.B., B.S. in 1886, M.D. in 1887, took the F.R.C.S. in 1888, and was elected F.R.C.P. in 1894.

Soon after becoming qualified Washbourn spent some time under Professor Grüber in Vienna and Professor von Baumgarten in Königsberg, chiefly in the study of bacteriology. He was appointed Demonstrator of Anatomy at Guy's in 1888, and Assistant Physician in 1890, when he also became joint Lecturer on Physiology, Demonstrator of Bacteriology, and Physician in Charge of the Electrical Department. A few years later, when the Lectureship on Bacteriology was established, he was appointed Lecturer. In 1889 he was elected Assistant Physician, and in 1892 Physician, to the London Fever Hospital. Subsequently he became full Physician to Guy's Hospital.

In February, 1900, he went to South Africa as Consulting Physician to the Imperial Yeomanry Hospital, which was just then being organised. For his services he was created a C.M.G., and in 1901 was gazetted Consulting Physician to His Majesty's Forces in South Africa. He returned to England in July of that year, and presided over the section of Pathology and Bacteriology at the Cheltenham meeting of the British Medical Association. During the winter he employed most of his spare time in the preparation of the Croonian Lectures on Acute Pneumonia, which he was to have delivered before the Royal College of Physicians. But in March he was attacked with influenza, to which for many years past he had been subject. This illness was followed by pleurisy with effusion, which unhappily proved to be of tuberculous origin. He died at Tunbridge Wells on June 22nd, within two days of completing his thirty-ninth year.

Although a man of wide knowledge and experience in general medicine, Washbourn's chief interest centred in the study of infectious diseases, for which his bacteriological training and his position at the London Fever Hospital eminently fitted him. Most of his work was done in this field. The Medical School at Guy's is largely indebted to him for the admirable Bacteriological Department that exists there now; and it was on his initiative that bacteriology was added to the medical curriculum by the College of Physicians. His most important research work was done in connection with the *diplococcus pneumoniæ*, especially in respect to its varieties and the methods by which their virulence may be estimated. In 1897 he published an account of an anti-pneumococcic serum which he was the first to prepare, and which it was ever his wish and hope might eventually be made of certain therapeutic value. How bacteriology may aid the study of epidemics is admirably shown by his report on the memorable epidemic of typhoid fever at Maidstone in 1897. He was called in to advise the local authorities, and his bacteriological examination of the various sources of the water-supply indicated the pollution of a certain one. On his recommendation this source was cut off, and the epidemic soon ceased. In the enquiry held subsequently by the Local Government Board, the Inspectors accepted his views as correct, in spite of strong and adverse expert evidence.

Washbourn also published, both alone and jointly, many papers on bacteriological and clinical subjects, of which the most important were upon diphtheria and its treatment;

infectious sarcomata in dogs; and dysentery and enteric fever in South Africa. He was the joint author of a text-book on infectious diseases.

Though still young in years at the time of his death, Washbourn had yet fully justified the expectations that were formed of him when he was still an undergraduate by his teachers and fellow-students. Perhaps his chief characteristic was tenacity of purpose. If he made up his mind to accomplish any particular design, he was not to be swerved from his purpose, but took the most direct path towards it that was consistent with honesty and fairness. At the same time he could, when necessary, exercise unlimited patience, and always took an infinite amount of pains. Being, as regards scientific work, of an extremely sceptical turn of mind, the experimental results of himself and his pupils were repeatedly and minutely tested; consequently, his work is characterised by a stability and thoroughness which is sadly wanting in much of the so-called scientific work of the present day. Enthusiastic in all his undertakings, he had the precious gift of being able to inspire those with whom he was brought in contact with some of his own enthusiasm. Hence he was an admirable teacher. For honest work he always gave full credit, and if a friend or pupil materially helped him in any production, he always insisted on its being put forward in their joint names. He was a fluent, lucid, and, when occasion served, a witty speaker. In outward appearance he seemed to be considerably older than he was. His intellect was keen; his judgment accurate, though, when necessary, quickly formed. Perhaps it was the combination of all these qualities that accounted for the wonderful power, which he certainly possessed, of inspiring confidence and of quickly making his personality felt by those around him. The writer will never forget an occasion when this trait was particularly evident. A medical man had been summoned before a magistrate by a local health authority for failing to notify a case of infectious disease, his failure so to do having given rise to disastrous results. His defence was that he had honestly made an error in diagnosis. This defence was good enough, but the unfortunate practitioner himself did his best to discredit it by the extremely bad manner in which he gave his evidence. He could not give even his own counsel, a well-known and able barrister, a straightforward answer; and all the facts, though favourable to himself, had to be dragged from him. A stranger listening to the proceed-

ings would have thought that the man was a hostile witness under cross-examination. The magistrate was evidently against him, and was taking copious notes; nor did his attitude appear to be affected by two or three medical witnesses who spoke for the defendant. But Washbourn, who was called to show that the error in diagnosis might honestly, though easily, have been made, had hardly been in the witness-box two minutes before it became quite clear that the magistrate's mind was undergoing a complete change. He shut up his note-book, made a remark or two to the effect that it was not necessary to pursue the matter further, and within a few minutes of Washbourn's appearance dismissed the case.

In private life Washbourn had a host of friends, by whom his advice was frequently sought—by no means always on professional questions—and never in vain, for he was always kind and sympathetic. He was fond of outdoor sports, though he had but little leisure during recent years to give to them. He was a great favourite with the students at Guy's, in whose work and amusements he took a hearty interest. He married in 1893, but his wife died a year later after childbirth, and he remained a widower. His child, a daughter, survives him.

He has gone, gone ere his prime, with his work in our Society, as elsewhere, unfinished. In many places, by many friends, he is missed and mourned; yet nowhere more than by those of us who have been accustomed to meet him at the various gatherings of the Epidemiological Society.

E. W. G.

The Ordinary Meetings of the Society are held at

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